ALPHA WAVE ENHANCEMENT AND THETA WAVE SUPPRESSION IN THE CONTROL OF EPILEPTIC SEIZURES

By

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In the romantic imagination of the public, scientific progress is pictured as a single individual stumbling into some discovery while toiling late and alone in a makeshift laboratory. In reality, scientific progress is the sum total of the tiny and painstaking contributions of generations of researchers. It seems fitting, therefore, that before beginning this report of my own small contribution to the therapeutics of epilepsy, I should acknowledge the encouragement, assistance, and cooperation of the many colleages without whom this work would not have been possible.

I am particularly grateful to the members of my committee: Drs. Hugh C. Davis, Cynthia Belar, Robert Isaacson, Warren J. Rice, and B. Joe Wilder. Dr. Davis, the chairman, stepped in unselfishly to fill the void left by the sudden death of Sidney Jourard. Dr. Belar loaned us the seemingly unloanable 5400 computer at a time when ours had given up the ghost. Dr. Wilder's talk at the Michigan Epilepsy Center in 1973 convinced me to come to the University of Florida. Dr. Isaacson kindly shared his profound professional expertise and his unerring eye for precision and detail. A special thanks to Dr. Warren Rice, my cochairman, who contributed time, effort, and understanding for which I can never fully express my gratitude.

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This project has been simultaneously frustrating, fascinating, exhausting and stimulating. I hope as well that it will serve as a stepping stone for other researchers exploring the new and intriguing field of physiological feedback.

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The present research was concerned with investigating the EEG physiological feedback technique of combined alpha wave enhancement, theta wave suppression, and its effect upon the attenuation of epileptic seizures. A review of the literature indicated a possible link between midrange synchronous EEG activity, chiefly in the alpha range, and inhibition of seizures. In addition, the review demonstrated a possible connection between slow wave hypersynchony, chiefly in the theta range, and seizure propagation in focal seizure patients.

Five subjects, four male and one female, were employed in the study. All subjects were classified as having uncontrolled focal seizures of at least five years' duration. Each subject was required to maintain a seizure diary one month prior to and throughout the course of the experiment. All subjects were administered EEGs using the training referents T_3-T_4, C_3-C_4 before training was begun and immediately after completion.

Following an A-B_1-C-B_2-(D) research design (A = baseline, B_1 = contingent feedback 1, C = noncontingent feedback, B_2 = contingent feedback 2, D = theta suppression), subjects were trained to enhance EEG alpha wave and suppress theta wave production.

The results indicated support for the hypothesis that enhancement of synchronous midfrequency activity had a significant effect upon seizure rate. Discrepancies between these results and those of Sterman were discussed in terms of homogeneity of seizure type. The greater variability of seizure classification of Sterman's subjects made his results difficult to interpret from an etiological standpoint. The present study's concentration only on focal seizure patients increases the specificity of results for this subgroup.

Methodological problems were discussed in terms of extending baseline data or interjecting a multiple baseline approach to more accurately gauge the effectiveness of training procedures. The extension of the noncontingent condition was recommended to permit a more direct assessment of treatment effects.

CHAPTER I
A REVIEW OF THE LITERATURE

The sky spun like a mighty wheel;
I saw the trees like drunkards reel
And a light flash sprang o'er my eyes,
Which saw no farther. He who dies
Can die no more than I died.

I felt the blackness come and go,
And strove to wake, but could not make
My sense climb up from below.

Lord Byron
Mazeppa

Do not believe that the gods cause illness and madness and do not hope to bring healing and help the sufferer by means of magic spells or false sorceries.

Hippocrates
The Sacred Disease

The great contribution of Hippocratic medicine was its introduction of an objective, naturalistic and empirical attitude to the biological--and hence the behavioral--sciences. As early as 400 B.C., Hippocrates had recognized the existence of epilepsy as a specific ailment (Esper, 1964, p. 119). Yet, even in Lord Byron's time--he himself was reputedly an epileptic--seizures were still being attributed to mystical causes rather than to any specific organic or functional disturbance. The word seizure was originally used to describe epileptic convulsions because the person was believed to have been seized by the devil.

Today these symptoms are known to be associated with abnormal electrical activity in the brain. In some cases, this abnormal

activity can be shown to have its origin in a specific lesion in the cortex. These cases are known as focal seizures. In many other cases, no specific lesion or focus can be illustrated. These are nonfocal epilepsies. The epileptic disorders have proven themselves to be stubborn adversaries of modern medical science, and as yet there are no generally reliable treatment modalities for the disabling and disturbing handicaps epilepsy creates.

Recently, there has been interest in research which applies the techniques of physiological feedback to the control of epileptic seizures. While the results of this research are inconclusive, and the entire effort has been fraught with controversy, the success that other workers have had using physiological feedback techniques to treat other clinical disturbances implies that this line of investigation ought to be examined carefully. This review of the literature is devoted to that end.

Definition of Epilepsy

It is important to note at the outset that epilepsy is not a specific disease entity, but is rather the name given to certain signs of central nervous system dysfunction. Penfield and Jasper (1954) have described these clinical indications as a "state produced by an abnormal excessive discharge within the central nervous system" (p. 22). Jasper, Ward and Pope (1969) define epilepsy as a "sudden paroxysmal discharge" (p. 10) and add to it the concept of paroxysmal dysrhythmia of the EEG and hypersynchrony to indicate not only excessive firing of individual cells, but also massive discharge of many neurons in unison, "abolishing the finely-organized tempero-spatial patterning characteristics of the normal integrative activity of the brain" (p. 11).

Inherent in this definition is the diagnosis of a seizure disorder via the abnormality of an individual's interictal EEG.

Epilepsy is often given as a diagnosis when clinical convulsions and EEG manifestations are present, and the observed disturbances are not secondary to an existing condition such as hypoglycemia, tumor, or infection. Essential to this definition is a chronic recurrence of symptoms.

Such a restriction is not entirely without reason, for although the seizure is merely a sign of central nervous system dysfunction, and thus has no one etiology or mechanism, the seizure in all cases does utilize common neurophysiological mechanisms during its evolution. The usual sequence of a seizure is characterized by aura, spread, generalization, and cessation. The major motor seizure begins with an abnormal electrical discharge in one part of the brain, spreads to other areas, generalizing to a complete disruption of normal sensory and motor activity, and then terminates (Jasper, 1969).

Medical Treatment of Epilepsy

While seizure activity may be the sign of some underlying neuropathogic or neurochemical dysfunction of the brain, control of the symptom alone is of immense benefit to the person with epilepsy and indeed is the current goal of medical therapy. It is estimated that approximately one-half of one percent of the population, or about twenty million people worldwide, suffer from such seizures.

No single treatment procedure is effective in the treatment of many epileptics. The condition is treated today principally through medication with anticonvulsant drugs. These drugs, however, fail to

reduce seizure activity in 20 to 30 percent of all patients (Coatsworth, 1971). Even the patients who are helped by the medications risk side effects that may be serious and debilitating. For example, phenytoin sodium (Dilantin), perhaps the most widely prescribed antiepileptic agent, is known to elicit some fifteen definite and eighteen possible side effects (Bray, 1959), and to provoke a possible permanent degeneration of cerebellar neurons (Selhourst, Kaufman and Horowitz, 1972; Julien and Halperin, 1971). Phenacemide (Phenurone), a drug used in particularly intractable cases, is highly toxic and can lead to such bizarre psychic manifestations as suicidal ideation, paranoia, and acute psychotic states (Livingston and Pauli, 1954).

Prior to the introduction of appropriate medication there was much interest in dietary cures for epilepsy, but these treatments were not effective. One dietary method was a dehydration regime pioneered by Dr. Fay in Philadelphia in the 1930's. The ketogenic diet was developed after investigators noted that seizures stopped shortly after the onset of a fast, but the results did not extend beyond the starvation period. One method involves starving and dehydrating the patient for a period of several days, then prescribing a diet that contains four times as much fat as carbohydrates and protein combined. The procedure works best with children. This treatment can adversely affect the blood vessels of adults and is extremely complicated to administer (Livingston, 1960).

Surgical intervention is indicated when medical treatment has proven ineffective and the results of the operation on the patient's brain will not alter functioning or cause debilitating symptoms more severe than the seizures themselves. Surgical treatment is only

possible if the lesion is accessible and its removal will not severely impair the functioning of the brain.

The forms of surgical intervention most commonly used are:

1. the excision of localized cortical areas exhibiting constant abnormal electrical discharge;
2. temporal lobectomy for patients with clinical psychomotor seizures associated with focal, temporal lobe electroencephalographic abnormalities; and
3. partial hemispherectomy in certain cases of epilepsy associated with unilateral cerebral atrophy (Livingston, 1960).

Surgery clearly is indicated for only a small number of epileptics, and the probability of application to the general population of epileptics is limited. Rasmussen (1975) reported that of 129 patients treated surgically for the removal of nontumoral lesions, 48 percent has a moderate to no reduction of seizure frequency. Thus, none of the conventional techniques for the treatment of epileptic disorders are completely satisfactory. Additionally some 30 percent of the total epileptic population is left without an effective therapy.

Learning-Theoretic Models of Epilepsy

The two main types of medical intervention described in the preceding section are all based on the assumption that the epilepsy is a sign of some physiopathology of the central nervous system. And, indeed, it is often the case that seizure activity is related to some specific lesion. But the origin of nonfocal seizures, where no lesion is known to exist, is a matter of controversy. While many tentative

hypotheses for nonfocal seizure activity postulate some form of neurophysiological abnormality, other investigators believe that " . . . environmental conditions external to the epileptogenic tissue may be a major source of potentiating or attenuating the seizure disorder . . ." (Mostofsky and Balaschak, 1977, p. 723). The above authors provide an extensive review of the behavioral studies dealing with the treatment of epilepsy. They organize the review into several paradigms consisting of: 1) reward management, 2) self management, and 3) phychophysiological procedures. Since physiological mechanisms provide a more fundamental intervention procedure related to the possible central mechanism to explain seizures, the remainder of this literature review will concentrate on phychophysiological procedures. It is this type of etiological thinking that provides a theoretical underpinning for the use of physiological feedback as a therapy in cases of uncontrolled epilepsy. The following section will examine theories of both types, beginning with the physiological approach.

Learning Theory Models

Since physiological explanations are usually employed in the explanation of epilepsy, several weaknesses occur when it is employed as a unitary explanation:

1. how does the patient's condition differ on the one day which he has a seizure as opposed to the five on which he has none; and
2. what accounts for spread--can it be explained adequately in physiological terms? (Rodin, 1968)

The origin of learning theory explanation of epilepsy was the discovery in 1960 that a normal brain could "learn" to develop an epileptogenic focus without any physical trauma to the cells (Mostofsky and Balaschak, 1977).[1] Morrell's work (1960) demonstrated the formation of a "mirror focus," or secondary epileptogenic lesion (SEL), in the hemisphere opposite the existing lesion. By Morrell's definition, a SEL is "an electrophysiologically defined area of paroxsymal discharge, at least one synapse removed from the primary zone, but connected with massive neural pathways" (pp. 538).

The mirror focus type of SEL that Morrell studied developed in two phases appearing in the hemisphere opposite the primary lesion in the homologous brain area. In the first stage, the mirror focus develops spikes only in response to spiking in the primary focus. In the later stage, the secondary focus becomes entirely independent, no longer requiring the primary focus to act as a trigger.

The SEL can be viewed in relation to the "kindling phenomenon" first reported by Goddard, McIntyre, and Leech (1969). Goddard undertook his research to study the possible epileptogenic outcome of a "chronic irritant" on cortical tissue. His theory arose from the observation that "some patients who seem to have made good recovery from known head injuries become epileptic at a later date" (p. 297).

Goddard's experiment involved the use of low level stimulation of the brain of his experimental rats with implanted electrodes. He found that subthreshold stimulation, inadequate to trigger a seizure on its own, would eventually produce a seizure after repeated

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[1] "Learning" is used loosely here to refer to changes in the electrical characteristics of brain tissue in the absence of known physical or chemical events preceding the change.

stimulation of the area. Subsequent stimulation would produce a seizure after repeated stimulation of the area, even months later. This phenomenon has also been observed by Racine, Gartner, and Burnham (1972), Tress and Herberg (1972) and Wada (1976).

Why is a learning-theoretic model invoked to explain these results? Goddard observed in his experiment that the optimum frequency at which the stimulation should be applied to produce an eventual seizure in the rat was one a day. Stimulating the brain more often deterred the development of eventual epileptic activity. It is hard to understand this in terms of direct tissue damage; moreover, the magnitude of the potential applied to the brain as a stimulus does not effect the time interval before the area becomes an epileptic focus. The neurons appear to exhibit a type of learning curve, reaching 100 percent accuracy when the area becomes an autonomous epileptic focus. The kindling phenomenon has also been observed to occur when subthreshold doses of seizure-inducing drugs such as pentylenetetrazol (Metrazol) are administered.

The learning-theoretic model offers an explanation for the development and spread of seizure activity. The immediate question, then, is whether the process can be made to work in reverse, that is, can the brain learn regulatory or pacemaker activity? To answer this question a discussion of normal brain wave patterns and their controlling mechanisms will be presented. Based upon these concepts, the possibility of applying EEG physiological feedback as a means of attenuating the evolution of paroxysmal activity will be explored.

One way of analyzing EEG activity is to divide it between synchronous and asynchronous patterns. Synchronous patterns consist

of steady, periodic activity which ultimately relate to the extent to which neuronal activity is simultaneously active. In EEGs it has been characterized by the coincidence of waves of similar shape. Though there have been no exact quantitative measurements of synchrony and its identification is usually based upon experience, it has been estimated that normal synchronous activity requires between 0.1 to 1.0 percent of a cell population firing to produce a 100uV wave on the surface (Diver and MacGillivary, 1977). This activity has been described by Chase and Harper (1971) as essentially cortical idling--the absence of any specific information processing activity. Normal synchrony has been most notably associated with alpha and slow-wave sleep.

Probably the best studied of human synchronous rhythms is the alpha rhythm (8-12Hz), whose role in relaxation processes has been lately popularized. The alpha frequency was discovered in 1929 by the German neurologist Hans Berger (Berger, 1929). He learned of the inhibitory nature of the alpha rhythm by observing that its production was blocked by directing attention toward a stimulus or event. Adrian and Matthews (1934) found that alpha is not always blocked by any form of attention; what is required is the perception of attempted perception of a pattern. They also found the difficulty of the task to which attention was directed was a factor in the degree of alpha blocking that occurred. Modern research on human occipital alpha has shown that this pattern is likely to be dominant when the oculomotor and visual systems are not activity tracking a target or attending to visual information.

Asynchronous patterns appear as low voltage fast waves, similar to random electrical activity and are associated with active attention

and the processing of information. These patterns in the frequency range above 13 Hz are identified as beta activity (Kiloh and Osselton, 1966).

Inhibitory Synchronous Activity and Seizure Production

Andersen and Andersson (1968) postulate that the cortex is almost totally dependent on the thalamus for its synchronous activity. According to their Facilitative Pacemaker Theory, all major thalamic nuclei have the capacity to generate rhythmic activity. Each nucleus functions as a small pacemaker, projecting to a specific cortical area. There may be as many as thirty to forty thousand of these pacemakers. Diver and MacGillivary (1977) describe the general principle underlying pacemaker activity in the generation of synchronous rhythms, particularly the alpha rhythm. They state that inhibitory activity is promoted by a series of feedback mechanisms that cause a synchronous inhibition through the action of a class of neurons, which they term recurrent inhibition. Eccles (1969) has characterized inhibition as "that action by a class of synapses that opposes excitation and tends to prevent the generation of impulses by excitatory synapses" (p. 235). Thatcher and John (1977), in speaking about inhibitory synchronous activity, posit an interactive loop process which keeps excitatory/inhibitory activity in bounds, and which functions to "prevent epilepsy and regulate the levels of excitation such as sleep and wakefulness" (p. 117). Thatcher and Purpura (1972) further argue that thalamocortical synchronization, particularly as it applies to the alpha rhythm, may be characterized as a "scanning mechanism" which alternately excites and inhibits cortical activity. According to Thatcher and John (1977)

a subcortical scanning pulse exists that monitors the state of neural excitability. They cite evoked potential research which demonstrated a phasic alteration in excitability corresponding to the frequency and phase of the alpha rhythm. During faster, desynchronous activity, usually coincident with active focusing of attention, the loop systems are broken down into a large number of smaller loops or independent oscillators parsing activity according to the specific system involved.

Hypersynchrony, considered by most the hallmark of seizure production, has been characterized by Jasper (1969) and Penfield and Jasper (1954) as having two main functional components: 1) existence of an excessive firing of individual cells and 2) the excessive synchronization of unit discharge activity which does away with the organized patterned electrical activity that is characteristic of normal brain function.

In discussing the mechanism of synchronization, Diver and MacGillivary (1977) state that the production of normal alpha activity is far different from the hypersynchronous patterns associated with seizures. The general lack of inhibitory mechanisms in the recruitment of adjacent neuronal areas is considered the prime mechanism of the genesis of hypersynchrony. If the interactive loop hypothesis of Thatcher and John is invoked, the excitatory/recruitment phase is not counterbalanced by the normal inhibitory one. In such a case, greater and greater portions of neuronal activity begin to respond without control.

Given the consensus that seizure activity is characterized by a lack of inhibitory synchronous activity, and in view of the concepts derived from the SEL and the kindling phenomenon, the technique of

physiological feedback may be an efficacious method of establishing an inhibitory augmentation mechanism. The remainder of this review will be aimed at further development of this concept, beginning with an overview of the topic area.

Physiological Feedback

Physiological feedback is a technique wherein the subject is given continuous information about the state of some physiological variable of which he is not normally aware, such as blood pressure or EEG activity.

As currently used, the technique involves the amplification of minute biological electrical potentials into signals large enough to record on a polygraph. The signals are then filtered to detect a certain frequency and voltage band to distinguish changes of a predetermined sort. The information is then fed back to the subject using devices such as tone generators, slide projectors, or indicator lamps. In this manner, the individual is given information about his physiological state in a useful form. Individuals have been taught with this method to exhibit a heightened degree of control over heart rate, blood pressure, galvanic skin response, muscle tension, skin temperature, and EEG alpha activity (Barber, DiCara, Kamiya, Miller, Shapiro and Stoyva, 1970-77; Miller, 1978).

Researchers have investigated the applicability of physiological feedback as a therapeutic intervention into a great variety of pathological states. Some conditions for which physiological feedback therapy seemed to yield promising results were muscle retraining after atrophy or paralysis (Andrews, 1964), chronic anxiety (Raskin, Johnson, and Rondestvedt, 1973), tension headache (Epstein, Hersen, and Hemphill,

1974; Budzinski, Stoyva, and Adler, 1970), heart rate control in patients with heart disease (Troyer, Twentyman, Gatchel and Lang, 1973; Weiss and Engle, 1971), and control of essential hypertension (Benson, Shapiro, Tursky and Schwartz, 1971).

In these studies various physiological measurements were used as the variables that the subjects were taught to control. In the studies on muscle retraining, anxiety, and tension headache, the variable was muscle tension as recorded on an electromyograph. For the insomnia study, persons were taught to augment the theta wave of the EEG. In heart rate and blood pressure studies, the subjects were taught to control those variables directly.

While a variety of work has been done in the treatment of clinical disorders through physiological feedback techniques, only recently has EEG physiological feedback been used to investigate pathological states (Mulholland and Benson, 1971). Prior to this time, most physiological work using EEG rhythms as feedback variables was devoted to studying normal processes, such as sleep, meditation, and relaxation (Jonas, 1973). There is a growing body of literature reporting attempts to treat epilepsy through physiological feedback techniques.

The Initial Use of Physiological Feedback in the Treatment of Epilepsy

Early experiments in conditioning approaches to the treatment of epilepsy differed from the methods of physiological feedback. In one early sample, Stevens (1962) and her colleagues (Stevens, Milstein, and Dodds, 1967) attempted to extinguish spiking and other abnormal epileptiform EEG patterns. A tone and mild electric shock were administered to the subject at the moment the abnormal wave form occurred. When the tone was discontinued, discrimination disappeared

in all but one subject. That person was still able to respond in the absence of the tone when there was a paroxysmal discharge.

Another model for learning-theoretic intervention was explored by Efron (1956, 1957) who utilized the model of classical conditioning. His work was dependent upon the existence of an unconditioned stimulus, an odor, in the case he studied, which could arrest the seizure activity of the patient. He was then able to condition a visual stimulus, a bracelet, to the dissolution of the aura.

In a series of studies, Forster and Booker (1964) and Foster (1967 and 1972) developed a method for treating epilepsy where the seizures are induced by sensory stimuli. A stimulus which usually evokes a seizure was used via an extinction or habituation procedure which is designed to weaken the ability of the stimulus to provoke a seizure. Mostofsky and Balaschak (1977) have suggested that rather than an extinction or habituation paradigm for the attenuation of sensory specific induced seizures, Forster's method tends more to shift attention away from the seizure stimuli and, therefore, act as a competing stimuli that does not evoke the paroxysmal response.

The groundwork for efforts using the principles of operant conditioning in the treatment of seizures was laid by Sterman, LoPresti, and Fairchild (1969) who reported that cats who had been taught to produce sensory-motor rhythm (SMR), which ranges in frequency between 12-16 Hz measured over the coronal gyrus, were unusually resistant to seizure-producing drugs. In cats trained to increase SMR activity, there was a significant delay between the administration of the drug pentylenetetrazol (Metrazol) and the onset of preseizure symptoms. In untrained cats, no such delay was noted; the administration of the drug

was followed by a much shortened interval between preseizure symptoms and the seizure itself.

A few years later, Sterman and a colleague produced the first report of a treatment of a human epileptic through this technique (Sterman and Friar, 1972). In this case they reinforced an 11-13 Hz rhythm, which they maintained was the analog of feline SMR. This represented a break with earlier research that considered the mu, or en arceau rhythm (7-11 Hz) to be the human correlate of the sensorimotor rhythm.

Sterman and Friar (1972) now claim that human SMR is a rhythm distinct from both the alpha and mu rhythms. The frequency that Sterman and his colleagues have been identifying as SMR is extremely difficult to detect, due to its low amplitude. While most cortical synchronous rhythms are in the range of 50 μV or larger, human SMR is approximately 5-15 μV. It has thus been difficult to verify the existence and properties of this rhythm in the human EEG.

Kaplan (1974) reported that the low amplitude SMR was discernable in human EEG's through the use of a dual filtering system. The signal was prefiltered through a conventional analog filter and then digitally filtered on a PDP-12 computer.

Kaplan (1974) pointed out that the system used by Sterman and associates was unable to distinguish SMR accurately and, therefore, there was no proof that it was indeed SMR that was being reinforced. She was especially critical of the fact that in none of Sterman's experiments was there any documentation that the levels of SMR were increased by the feedback training sessions. In her experiment there could be no doubt about which rhythms were being reinforced.

Three dependent variables were measured in the Kaplan (1974) study. They were evaluations of clinical EEG's, seizure incidence, and power spectra (fast Fourier transforms of EEG epochs). The experiment involved the training of two epileptic patients with 12-14 Hz biofeedback for three months. No decrease in seizure activity resulted, and one of the patients dropped out of the study. The remaining patient and two others were then trained in the production of the alpha frequency.

Necessary changes in the medication that the one patient in both stages of the experiment received made it impossible to compare that subject's results directly. It is believed that the observed changes in both seizure incidence and power spectra for this subject are due to the medication changes.

Kaplan's other two patients, trained only in the theta-alpha production (6-12 Hz), showed a significant reduction in seizure incidence. However, no changes in the power spectra of the EEG's were evident. Thus, the clinical changes were not ascribed to learning of EEG synchrony. Rather, Kaplan attributed the changes in the subjects' learning to function on a lowered level of arousal. That two of the subjects later reported an increase of seizure incidence under stress was taken as a further indication of this interpretation.

In a later paper, Kaplan (1975) reiterated her criticisms of the methodology employed by Sterman. She emphasized in particular the impossibility of ascribing the observed clinical changes to changes in the synchronous rhythm production if the fact that these rhythms were indeed being produced was not demonstrable.

Gastaut (1975), the discoverer of the mu rhythm, agreed with Kaplan's criticisms and maintained that there was in fact no rigorous evidence that biofeedback training was ever effective in treating clinical disorders.

Recent Experimentation

In the last few years, several researchers have attempted to answer some of the questions and controversies surrounding this earlier research. Table 1 presents a summary of selected EEG physiological feedback studies in epilepsy. This more recent research has tested the effectiveness of alpha and theta rhythm training as well as the human SMR and has experimented as well with the reinforcement of combinations of synchronous activities of different frequencies. The results of this research are also equivocal and leave the issue far from settled.

Cabral and Scott (1976) contrasted the effects of alpha training with a Jacobsonian relaxation technique on seizure reduction. Their conclusion was that alpha training had a more consistent effect on seizure reduction.

Kuhlman and Allison (1977) designed a study in which EEG training was used to effect significant seizure reduction in three out of five patients. The study was designed to permit the determination of the actual changes in the EEG produced. Prior to the initiation of training to reduce seizures, three normal subjects were successfully trained to enhance the central mu rhythm in the 9-11 Hz band using 50 20-minute sessions. Similarly, three normal subjects given the same amount of training were unable to increase 12-14 Hz activity with

Table 1. Summary of EEG Feedback Studies in Epilepsy.

Study	Frequency Parameters	Subjects	Design and Training	Duration
Sterman & Friar (1972)	11-13 Hz (+)	1	Single case	3 mo.
Sterman, Macdonald, & Stone (1974)	12-14 Hz (+)	3	Group Pre-Post	6-18 mo
Finley, Smith, & Etherton (1975) and Finley (1977)	11-13 Hz (+) <10 (-)	2	Two single cases	10-22 mo.
Seifert & Lubar (1975) and Lubar & Bahler (1976)	12-14 Hz (+) 4- 7 Hz (-)	8	Group Pre-Post	6-9 mo.
Kaplan (1975)	12-14 Hz (+) 6-12 Hz (+)	2 3	Group Pre-Post	3-4 mo. 5-6 Mo.
Kuhlman & Allison (1976)	9-14 Hz (+)	5	A-B	1-2 mo.
Wyler, Lockard, & Inch (1976)	9-14 Hz (+)	5	A-B-A	1.5-6 mo.
Sterman and Macdonald (1977)	12-15 Hz (+) 18-23 Hz (+) 6- 9 Hz (-)	3	A-B-A-B	12 mo.
Sterman and Macdonald (1978)	12-15 Hz (+) 18-23 Hz (+) 6- 9 Hz (-)	8	A-B-A-B	12 mo.

feedback training. When training to attenuate seizures was instituted, none of the successful patients increased activity in the 12-14 Hz range but did enhance significantly production in the 9-11 Hz range.

This same study observed that occipital alpha increased in the successful cases. They state that "in fact, the increase in the occipital alpha frequency is a virtual mirror image of the seizure frequency" (p. 122).

One further implication of these results is that seizure reduction is not dependent on alteration of a specific EEG pattern, or the increase of any specific inhibitory cortical rhythms. Research by Quy (1977) on three chronic seizure patients led him to the conclusion that indeed the most effective form of biofeedback training for these patients was one which reinforced all synchronous activity in a broad, midrange spectrum.

A number of investigations have borne out this role of the mid-range spectrum in seizure reduction. In particular, the suppression of the slow theta frequencies as well as the enhancement of the midrange spectrum seems to bolster the effectiveness of the treatment. This result has been reported by Finley, Smith and Etherton (1975), Lubar and Bahler (1976), Wyler, Lockard, and Inch (1976), and Sterman and Macdonald (1978).

Budzinski and Stoyva (1969) reported that when frontal EMG levels became particularly low, the subjects tended to display an increase in EEG theta waves. Sittenfeld, Budzinski, and Stoyva (1976) demonstrated that profound muscular relaxation was associated with the appearance of theta in the EEG. Budzinski, in "Clinical Implications of Electromyographic Training" (1977, p. 438), reported that at the lower levels of frontalis muscle tension there arose an an inverse relationship with EEG theta.

Beatty, Greenberg, Deibler, and O'Hanlon (1974) reported that the absence of theta (which they defined as 3-7 Hz) was particularly associated with the ability to maintain vigilance. O'Hanlon and Beatty (1975) showed that theta suppression may prevent or lessen the performance decrements typically associated with long, exacting vigilance tasks.

The link between theta rhythm and epileptic seizures has been firmly established. Gibbs, Gibbs, and Lennox (1937), in a study of 400 epileptic patients, noted the tendency of seizures to occur when the patient is awakening or falling asleep. They believe pre- and post-sleep stages modify the rate of normal cortical rhythm, placing a strain on the self-regulatory mechanism of epileptics with which they are unable to cope.

Gibbs, Fuster, and Gibbs (1948) and Jasper, Pertuiset, and Flanigin (1951) have confirmed the focal cortical origin of theta rhythms in a majority of epileptic patients.

In 1974, Johnson and Meyer devised a phased biofeedback treatment procedure for an eighteen-year-old female epileptic who was allergic to Dilantin. The treatment was administered in a phased sequence beginning with alpha feedback, followed by alpha and theta feedback, and concluding with theta feedback alone, in an attempt to establish a low arousal antistress response. The patient, though unable to stop a seizure once begun, was able to prevent the onset of seizures by going into an alpha state when she felt an aura developing. At the end of one year she had experienced a 46 percent decrease in seizures.

Sterman's most recent study (1978) was designed specifically to investigate the importance of specific training frequencies in the

interpretation of EEG data. In his double blind study, patients were alternately trained to produce EEG activity in one of three frequency ranges. One was always the 6-9 Hz theta frequency, and the other was either 12-15 Hz or 18-23 Hz. Patients were rewarded for producing one in the absence of the other. After three months the contingencies were reversed without the patient's knowledge. For six of the eight subjects, significant seizure reduction occurred when the higher frequency was trained and the lower suppressed. When the higher frequency was within the 12-18 Hz band, the beneficial effects survived the reversal. For the six patients with seizure reduction, the average reduction in rate of seizures was 74 percent. Sterman interprets this as illustrating the specific role of training for various frequencies and rejects the claims by some detractors that the beneficial effects are an artifact of the design, produced by the patient sitting motionless and functioning at a lower level of arousal.

Summary and Hypotheses

Epilepsy is the term applied to a complex set of central nervous system dysfunctions with electrical and most often behavioral consequences. A further delineation of this syndrome would characterize it as an excessive firing of many neurons in unison, hypersynchrony, and slow wave activity, particularly in the theta range. This has been considered the hallmark of seizure initiation and propagation. Complex partial seizure activity is associated with abnormal EEG activity which generally begins in a localized area and generalizes as the seizure develops.

As conventional therapies for seizures of this sort are unable to effect significant improvement in 20-30 percent of epileptics, interest in the possibility of alternative treatment modes by eliciting learned changes in the brain's electrical activity became more important.

This interest was spurred by observations, which seemed to indicate that learning processes might, in some cases, be involved in the development of seizure activity. Morrell's discovery of the mirror focus phenomenon and Goddard's observation of the kindling phenomenon are the two principal pieces of research in this area.

The techniques of biofeedback, which have been used with success in the treatment of a great variety of other clinical entities, were used to train patients in the production of these cortical rhythms.

Initially a great deal of interest was focused on those frequencies believed to be the human analog of the SMR in cats. Feline SMR was known to be associated with the suppression of motion and was also observed to be correlated with resistance to seizures.

Further research has cast doubts on the specificity of the SMR as a factor in seizure reduction. Criticisms of some of these early studies, especially by Sterman et al. (1974), pointed out the importance of illustrating changes in the EEG as well as proving seizure reduction if any notion of cause and effect is to be developed.

It is the learning theory of seizure propagation that leads to the physiological feedback method of therapy. There is considerable evidence to suggest that brain cells can learn to be epileptogenic, either through mirror focus mechanisms, Morrell's secondary epileptogenic lesion, or through the Goddard kindling phenomenon. If the

brain can learn to be more easily excitable, can it conversely be made to learn inhibitory or pacemaker activity? This is what physiological feedback techniques attempt to accomplish.

In order to be effective, however, considerably more information on the precise mechanisms involved must be developed through research. Even if one accepts the learning theory, what waveforms should be reinforced? Sterman and his colleagues use a waveform assumed to be the human counterpart of the cat SMR and have claimed success with a small patient population. Additional study, however, has convinced the same researchers that SMR in humans exists as a rhythm distinct from either alpha or mu. Kaplan assumed that previous experiments were using a filter system which could not discriminate signals in the appropriate range adequately. Using more sophisticated electronic equipment, she established that the Sterman rhythm, when used in biofeedback training, did not work. Using low frequency synchrony was also inconclusive.

The literature, therefore, appears to indicate that biofeedback techniques are promising avenues to explore in the treatment of epilepsy, yet considerable fundamental research is needed before understanding and effective therapy can be accomplished. The literature dealing with seizure initiation and propagation points to inhibitory synchronous rhythms, especially the alpha rhythm, as a means of attenuating this activity. Enhancing the percentage of time the patient produces alpha while simultaneously diminishing the effects of hypersynchrony via learned suppression of slow wave theta may well provide a direct and parsimonious means of controlling seizure activity.

The hypotheses to be tested are as follows:

1. There will be a significant difference between pre- and post-EEG measurements. This difference will be manifested by a diminution of slow wave activity, chiefly in the theta (3-8 Hz) and delta (1-3 Hz) range, and an increase in the percentage of time in the faster range, particularly the alpha (9-13 Hz) range.

2. Seizure incidence will diminish over time, as a function of training, being significantly less at the completion of the study.

3. The time series measuring the rate of alpha production during and following intervention can be represented by a significant (positive) change in both level and slope as compared with baseline measures.

4. The time series measuring the rate of theta suppression during and following intervention can be represented by a significant negative change in level and slope as compared with baseline measures.

CHAPTER II
METHOD

The basic operations required to investigate these hypotheses require:

1) measures of concurrent dependent variables of alpha percent, average frequency, and average amplitude, theta percent and theta count for each subject over a sufficiently long time course to permit an evaluation of baseline, treatment, noncontingent treatment;

2) a measure of seizure activity via a diary begun before treatment and kept throughout; and

3) an independent measure of EEG frequency changes pre- and post-feedback training.

Subject Selection

Six subjects were selected using the following three main criteria: seizure history, frequency of seizures, and subject's being refractory to anticonvulsant medication. First, it was required that each subject have a positive history of essentially uncontrolled seizures for at least five years prior to the initiation of training. They were identified via patient records at the Veterans Medical Center, Gainesville, Florida, and through referrals from the Jacksonville Epilepsy Foundation and subsequently through medical records of the patient's neurologist. The second requirement was that following long-term anticonvulsant therapy with good compliance, the seizure

condition was not under adequate control. Finally, the requirement was established that the subject must be experiencing at least five seizures per month that were verified by family members, friends, or medical personnel.

Beginning approximately one month prior to the initiation of training and continuing throughout training, each subject was supplied with a seizure diary on which he indicated by day and time the occurrence of seizures. In each case family members or close friends were asked to monitor this record to ensure its accuracy. Subjects were frequently reminded of the importance of this diary and asked to bring it in weekly to assure understanding and compliance.

The following section presents Individual Subject Profiles indicating summaries of each subject's background medications, EEG data and pertinent medical and behavioral information.

Individual Subject Profiles

Subject #1 was a fifty-four-year-old divorced white male with a history of seizures from age thirty-one. He has been diagnosed as having complex-partial seizures with a right temporal parietal focus. The patient has been operated on with right parietal lobe surgery as a result of an old trauma. Medication prior to and throughout training consisted of Dilantin 300 mg. a day.

A review of EEG records revealed an asymmetry of fast activity because of high amplitude over the right parietal and temporal regions. Also, continuously low voltage and irregular slow waves in the theta and delta range were seen over the right parietal and temporal regions. This was considered an abnormal EEG because of the above. These

alterations were considered compatible with a structural lesion. The asymmetry and lateralized fast activity were thought to be due to the skull defect.

The patient's typical seizure pattern consisted of tactile sensory loss accompanying partial hand and facial paralysis on left side and an expressive aphasia ranging from markedly slurred speech to no speech at all. There was no loss of consciousness during these episodes.

Seizure frequency at the beginning of the study: 59 per month.

Subject #2 was a twenty-three-year-old single white male with history of seizures from age ten. He has been diagnosed as having psychomotor seizures with an unknown etiology. Medications prior to and throughout the course of training consisted of: Tegretol, 600 mg. a day; Tranxene, 7.5 mg., five times a day.

A review of the patient's history indicated some behavioral problems as a child, angry episodes, acting-out. These behaviors have not been noted in the patient's record since 1971.

Review of EEG records revealed an initial abnormal EEG with left-sided predominance; sharp activity was seen over both midtemporal and posterior temporal areas. Later records indicated a diffuse bitemporal slowing with background rhythms almost continuously irregular in the 2-1/2 to 5 Hz range over the temporal, parietal, and occipital regions. The latest clinical interpretation commented that little if any trace of alpha rhythm was evident. The patient's diagnosis has uniformly been psychomotor seizure presently with a bitemporal focus.

The patient experiences an aura with a "scary" feeling beginning in his stomach and feeling of pressure in his head. A typical seizure

includes lip smacking and finger movements. There is a post-ictal stupor and tiredness that lasts from several minutes to several hours.

Seizure frequency at beginning of study: 27 per month.

Subject #3 was a thirty-two-year-old white married male with a history of seizures, post traumatic in etiology, since 1975. Medications at the outset of training consisted of Dilantin 400 mg. a day and Tegretol 1000 mg. a day. During the course of training Celontin 500 mg. was added.

Review of EEG records revealed an abnormal EEG with focus of epileptiform discharge in the right anterior temporal regions. Throughout the tracing a focus of epileptiform sharp waves and spikes and slow waves were identified emanating from the right anterior temporal regions.

Behaviorally the patient has been described by his neurologist as having a decreased libido, a seemingly growing disinterest in sexual relations with his wife though no reported inability to achieve and maintain an erection, and a significant weight loss. The patient has been described as being depressed due to his inability to maintain his former lifestyle as worker and provider.

A typical seizure for this patient manifested itself with amnesia and automatic auras of short and mild duration as to be not useful in self-control. He also manifested various kinds of situational automatisms. This subject attended the study for three baseline days and five days of treatment trials but decided to drop out of the experiment.

Seizure frequency at beginning of study: 20 per month.

Subject #4 was a twenty-four-year-old white divorced female with a history of seizures since age nineteen. She has been described

as having psychomotor seizures of traumatic origin. The patient suffered a closed head injury in an auto accident two years prior to the onset of seizures. Medication at the outset and during the course of training consisted of Dilantin 100 mg., five times a day.

Review of EEG records revealed an abnormal EEG with epileptiform discharges in the right posterior temporal region. There was significant slowing into the theta and delta range over these areas and the fronto-temporal area.

Behaviorally the subject presented herself as the one patient most positive and interested in maintaining a normal lifestyle which included work and family. Even when the frequency of her seizures made it impossible to work, she continued to plan for the time when she would again be able to work. Despite large difficulties in her personal life, the patient was able to complete training with few disruptions.

A typical seizure for this subject consisted of a feeling of paralysis, a severe memory disruption, and often a feeling of disorientation and anger postictally.

Seizure frequency at beginning of study: 207 per month.

Subject #5 was a twenty-six-year-old white married male who was injured and sustained a subdural hematoma in 1969. The onset of seizures was in 1971 with frequent major convulsions and brief complex partial automatisms. Medication prior to and throughout the course of training were Mysoline 1000 mg. a day and Dilantin 300 mg. a day.

A review of EEG records revealed an initial abnormal EEG with the primary focus over the right temporal area. Subsequent recordings

indicated a markedly abnormal tracing with the occurrence of bi-synchronous generalized epileptiform activity maximal over the right hemisphere. The patient's present diagnosis is a complex partial seizure disorder with a right temporal focus with a secondary bi-lateral synchrony. There is some generalization of seizures to the grand mal type.

Seizure frequency at beginning of study: 19 per month.

Subject #6 was a twenty-four-year-old white single male with a history of seizures from age twelve. He has been diagnosed as having psychomotor seizures with a left-temporal focus secondary to two surgical procedures in the left fronto-temporal area for obliteration of an AV malformation in the middle cerebral circuit. Medications prior to and throughout the course of training consisted of Dilantin 500 mg. a day and Celonten 1200 mg. a day.

A review of the patient's history indicated a psychiatric hospitalization for an acute psychotic episode. Presently he is attending a day hospital program and is well motivated to seek employment.

A review of EEG records revealed an abnormal EEG with a disorganized rhythm with voltage in the range of 6-8 Hz. There was considerable slowing in the delta range with a discrete spike focus in the left midtemporal area (seen over the T_3 electrode) and considerable theta activity also. The diagnosis was a psychomotor seizure condition with a primary left-temporal focus.

The patient experienced an aura, a peculiar feeling in his chest, and a salty taste in his mouth. His typical seizure was like a dream of falling, temporary expressive aphasia and loss of train of thought.

Seizure frequency at beginning of study: 147 per month.

Procedure

Prior to the initiation of training each patient was administered an EEG using bipolar referents T_3-T_4, C_3-C_4 with an ear ground (using the 10-20 electrode placement system). The C_3-C_4 placement was collected on channel 1 and T_3-T_4 was collected on channel 2. Twenty to fifty minutes of recording was obtained for each subject. They were instructed to stay awake throughout the recording sessions but asked to keep eyes closed. Though every attempt was made to instruct subjects to remain awake, one subject, #1, did fall asleep during the post session. The light sleep portion of his record was omitted from the analysis. The record was obtained on paper and FM tape. This procedure was again followed immediately upon the completion of training.

During the preparatory phase each patient was individually interviewed using Mostofsky and Balaschak's (1977) seizure questionnaire and given a copy of a short article entitled An Introduction to Biofeedback (Fuller, G. and Sempell, P., 1977), a layman's guide to physiological feedback. This article was the basis for subsequent discussions of the nature of physiological feedback. A tape recording instructing the patient in proper procedure and providing samples of the kind of feedback sounds they might expect was played four times (see Appendix A for script)--once during the initial interview and prior to sessions 4-5 and 6--so as to familiarize them with this feedback mode. It was constructed to duplicate auditory feedback of 20 percent, 50 percent, and 100 percent success in producing alpha. They also were provided feedback for theta (alarm) and that of artifact from too much movement. Subjects were instructed to increase the occurrence of one tone and suppress the alarm. All subjects were instructed to keep their eyes closed. On a chalkboard in the feedback room a diagram showing the various frequencies and their respective feedback was provided to enable

the patient to grasp more concretely the nature and rationale behind the desired response.

Training took place from three to four times per week with a session, including electrode placement and posttraining feedback, lasting one and one-half hours. Standard Grass Disc Electrodes were used for recording. Placement was bipolar, using T_3-T_4 and C_3-C_4 for alpha enhancement and theta suppression, respectively, and an ear ground. Hewlett-Packard Redux electrolyte paste was employed and the electrodes were cemented to the scalp with collodion and dried with a compressed air stream. The criterion for acceptable electrode contact was less than 10,000 ohms resistance as measured on a digital volt-ohm meter.

Patients were seated in a comfortable, reclining chair, in a quiet room 15 x 13 feet. The room was further subdivided and darkened via the use of two cushioned standing partitions that served to militate against any extraneous light and sound and give the feedback space an isolated smaller appearance. To the rear and approximately three feet away sat the experimenter who monitored all patient behaviors and all feedback through a common headphone setup.

Feedback consisted of an auditory tone that varied in pitch (signifying frequency changes in the pass band) and loudness (indicating EEG amplitude within the pass band) whenever the subject produced six successive zero crossings in the alpha range (9-13 Hz). Also there was an alarm that sounded whenever his EEG frequency exhibited six successive zero crossings in the theta range (3-8 Hz) and remained on until such time that he ceased to produce waves in this range.

Experimental Design

Using an A-B_1-C-B_2(D) design, each patient was run through between 32 and 36 fifty-minute trials. Figure 1 indicates

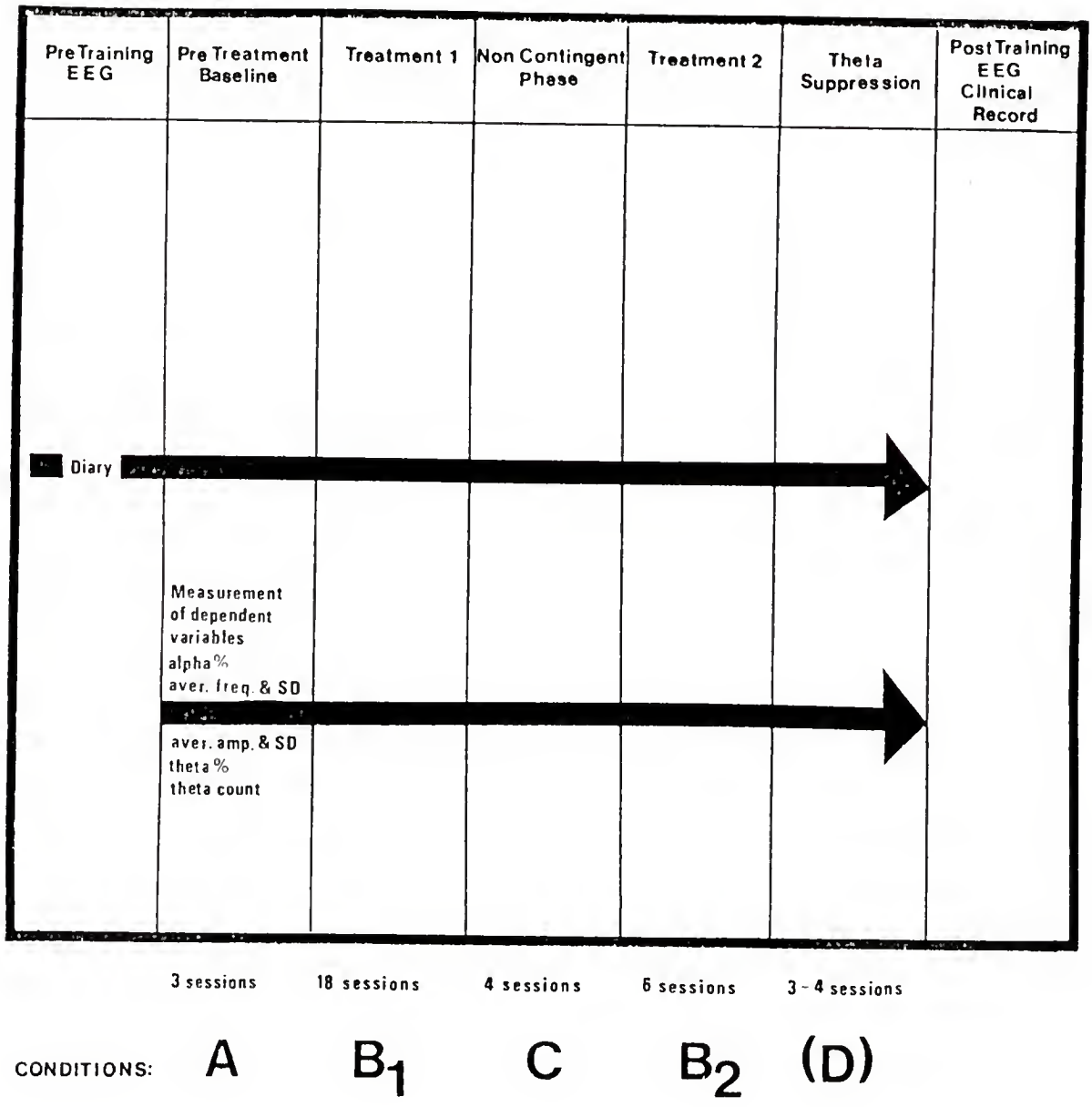


Figure 1. Schematic representing sequential operations of treatment conditions.

the various operations and measurements over the entire study for each subject. Each trial was divided into ten five-minute segments (see Figure 2) with a 45-second lag time when no data were collected. This lag time enabled the alpha-numeric component of the 5400 system to print out the previous segments data. At the beginning of this intermission the patient was required to respond to a light touch on the arm by lifting the index finger on the right hand. The patient was provided with the digital feedback obtained consisting of alpha wave percentage and theta wave percentage. Supplemental encouragement was given to enhance the alpha level and suppress the theta by keeping the sound on and the alarm off.

The initial three trials (Condition A) were baseline sessions when no feedback was provided. From trial four through twenty-two (Condition B_1), contingent feedback was provided for segments two through nine. Segments one and ten were again baseline where the subject was not provided with auditory feedback. Sessions 23-26 (Condition C) consisted of the identical ten-segment procedure but utilized noncontingent feedback for the eight feedback segments. Tape recordings of each subject's sessions 21 and 22 were interfaced with the feedback apparatus. Sessions 23 and 25 utilized the tape recording from session 21 and sessions 24 and 26 were based upon session 22. All procedures were identical to contingent trials including the usage of the digital feedback from these previous sessions. Upon debriefing at the conclusion of the study, it was clear that none of the patients were aware of this manipulation as the tape recorder had been used various times in the past during sessions to accustom the patients to its presence.

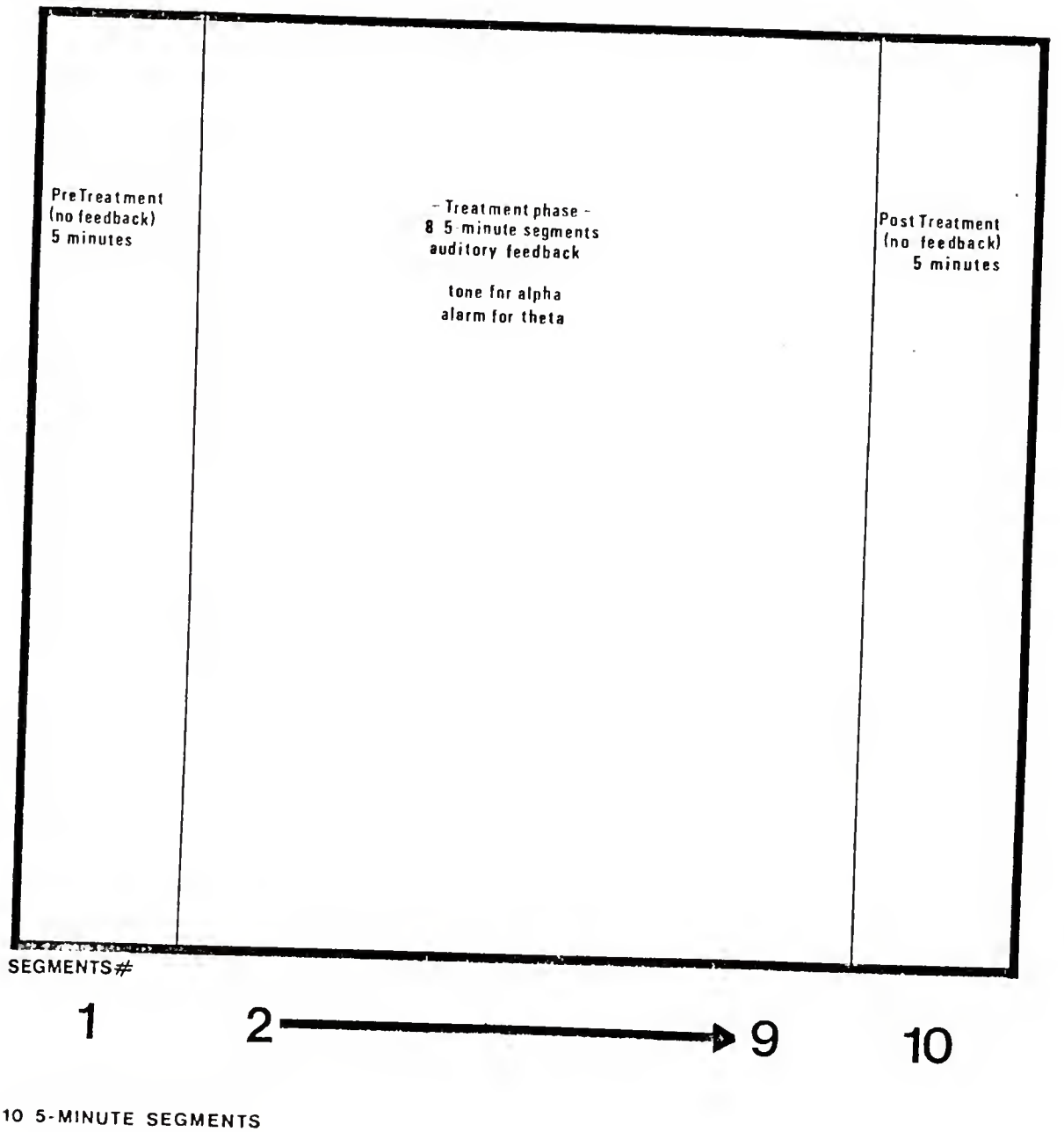


Figure 2. Schematic representing typical treatment trial with ten five-minute segments.

Sessions 27-32 (Condition B_2) were again contingent trials when feedback was provided depending upon the subject's alpha wave production and theta suppression.

For three of the subjects (#2, #5, and #6) C_3-C_4 location was employed as the active locus for feedback (Condition D), and the subjects began receiving the analogue tone indicating only theta's presence. They were told that the presence of the tone signaled theta and were instructed to keep the sound off. Subjects #2 and #5 received four such trials while #6 received three theta suppression trials.

Apparatus

The apparatus consisted of several major components: two EEG waveform analyzers (an Autogen 120a and Autogen 70a), an Autogen 5400 data acquisition unit, and a tape recorder. Both the 120a and 70a operate as variable bandpass filters for the EEG within the range from 2-20 Hz. The Autogen 120a permits feedback for EEG frequency and voltage levels continuous within its range of 2-20 Hz and 10-150 μV. The 120a was set at 100 μV so as not to reinforce epileptiform EEG activity. The output of the Autogen 120a consists of frequency, amplitude, percent time in a pass band, and the output of the 70a of discrete occurrences of a selected frequency within a pass band from which both a percent time and a count measure was obtained. The 120a was used to measure alpha (9-13 Hz) along the three parameters specified above and provide an auditory feedback, varying in pitch and loudness when the patient was producing EEG frequencies within

this range. Pitch varied with EEG within the pass band while loudness varied with EEG amplitude shifts. A distinctive alarm sounded when the subject's EEG frequency fell 1 Hz or more below the lowest frequency in the pass band and came on at 8 Hz. This alarm served two purposes: 1) to warn the subject of potential drowsiness and 2) to serve as a theta indicator. The Autogen 70a was used to record the percent time of theta and the number of discrete occurrences in the five-minute period. The Autogen 120a monitored from T_3-T_4 leads while the 70a monitored from C_3-C_4 leads. This was reversed only in the last condition (D).

The Autogen 5400 and the attached Alpha-Numeric Printer were employed to tabulate the data from the 120a and 70a and produce a paper tape recording that was timed to print out a data summary every five minutes. In addition to the means of each of the variables it provided standard deviations of alpha amplitude and average frequency standard deviations between 2-20 Hz.

The Akai tape recorder was interfaced with the feedback units to allow for tape recording and playback capabilities during the noncontingent trials.

Data Analysis

The two channels of EEG data collected were obtained, before and after physiological feedback training, both on chart paper and FM tape, C_3-C_4, channel 1, and T_3-T_4, channel 2, (International 10-20 electrode system). They were analyzed via the method of frequency analysis. The program, run on a PDP-8E computer, was designed to analyze results between the frequencies 1-20 Hz in 0.5 Hz increments.

The input from the FM tape was prefiltered with a Krohn-Hite filter set between 1-20 Hz to exclude frequency artifact. The program sampled the input 1000 times/second and timed each zero crossing, storing each in a register. At the end of a ten-minute epoch, input was terminated, and the contents of each register were printed. It then measured the time elapsed since the last data input and formed a histogram indicating the number of counts in each of the 0.5 Hz period bins. In addition, the average frequency within each of seven bands (1-3 Hz, 3.01-5.99....18.1-20 Hz) together with the percentage of total time within each of the pass bands, was tabulated and printed. The values of these bands were selected to approximate the ranges of delta, theta, and alpha frequencies in three Hertz increments. To produce a meaningful frequency axis, the data were collected in time intervals such that their inverse was close to the integer frequencies and their midpoints (i.e., 1, 1.5, 2, 2.5, etc.).

A Chi-square test was performed to examine the two histograms to test the hypothesis that the EEG data were drawn from different populations. Further, the individual Chi-squares were obtained in approximately 3 Hz bands to determine where significant effects took place within the 1-20 Hz population.

Seizure diary data were examined as a function of treatment conditions to determine the possible correlation of seizure rate with the acquisition of the dependent variables. In addition, a correlation between seizure rate and all dependent variables was performed.

The statistical analysis was directed to determining the significance of changes between the separate conditions of the experiment, $A\text{-}B_1\text{-}C\text{-}B_2\text{-}(D)$, in the sequence of treatment shifts. The output from the Autogen 5400 was punched onto cards and subjected to a time-series

analysis using an autocorrelation function and curve-fitting procedure. The data in the study represented a 7 x 1761 matrix (each of the depenend variables times each of ten five-minute epochs for each of the thirty-six trials). An autocorrelation function is a correlation of a time-series with itself. It is obtained by pairing observations of x units apart (Gottman, McFall and Barnett, 1969). This then provided the serial correlation of these data as a function of lag. For the baseline period lags of one through ten were employed, while for the pre- and post-periods lags of one were employed. The curvefitting procedure involved fitting the data to the least squares straight lines. The difference between slopes and between means was then calculated using as a test of significance a series of all possible t-tests.

Changes affected by the treatment procedures were analyzed by three procedures: 1) slopes within treatment conditions were assessed with respect to whether each slope was negative, zero, or positive; 2) differences between slopes within each period for each variable and, finally, 3) the differences between the means of all baseline-treatment combinations within each subject were calculated.

The presence of a slope significantly different from zero within a condition indicted that a relationship existed between the treatment conditions and the EEG variable under consideration. For example, a significant positive slope demonstrated that a direct relationship existed between application of the treatment and an increase in the EEG variable. Likewise, a negative slope demonstrated that the EEG variable decreased in magnitude over the course of the treatment condition.

The significance of the slopes was evaluated following the removal of the autocorrelation effects. This removal of the autocorrelation was made necessary since EEG data collected on a subject was predictive of data produced over successive periods. Once accomplished, the data assured the independence of repeated observations over time. Following

this, a least squares line was determined for the EEG data within each subject, for each variable during each condition.

The autocorrelation and least squares analysis was accomplished by an evaluation of the data with a commercially available autoregression program (Barr, Goodnight, Soll, and Helwig, 1976). The program's output provided ordinary least squares analysis, autocorrelation statistics and estimates of the least squares statistics after removal of autocorrelation, together with the calculation of the significance levels of the tests.

Next, selected contrasts between slopes were calculated using a modified "t" statistic. The difference was calculated by the following equation:

$$t = \frac{\hat{x}_1 - \hat{x}_2}{\frac{n_1 s_1^2 + n_2 s_2^2}{n_1 + n_2 - 2} \quad \frac{1}{n_1} + \frac{1}{n_2}} \qquad (1)$$

where $\hat{x}_1$ = baseline slope estimate

$\hat{x}_2$ = treatment slope estimate

n_1 = number of observations in baseline condition

n_2 = number of observations in treatment condition

s_1^2 = baseline slope variance

s_2^2 = treatment slope variance

The significance was evaluated to allow for adjustment of alpha error as a function of multiple pairwise contrasts using t-statistics (Games, 1977).

Finally, selected contrasts between the means within each condition were accomplished. The equation was the same as (1) above but employed the estimated mean condition value for $\hat{x}_1$ & $\hat{x}_2$, and s_1^2 & s_2^2 terms were replaced by the error sum of squares obtained from the autoregression program. Again, the table provided by Games (1977) was employed to evaluate significance levels.

CHAPTER III
RESULTS

The results are detailed in three major sections: 1) hypothesis 1, frequency analysis data of pre-/post-EEG measures; 2) hypotheses 3 and 4, time-series analysis of day-by-day training data comprised of alpha and theta changes; 3) hypothesis 2, seizure diary data and its relation to treatment conditions.

Data with Respect to Hypothesis 1

Changes in the Frequency of EEG Occurrence <8 Hz and in the 9.02-12.05 Hz Range

This hypothesis predicted that subjects will display a frequency shift from slow frequency ranges <8 Hz toward predominantly alpha activity in the 9-13 Hz range. This result was predicted from the contingencies presented in the experiments, namely, a) physiological feedback and instructions to suppress low frequency activity in the <8 Hz range, and b) instructions to increase the occurrence of frequencies between 9-13 Hz (exclusive). (See Tables 2-11 for chi-squares, number of counts and mean frequency data for all subjects.)

Subjects #4, #5, and #6 displayed a frequency shift from low frequency activity toward faster as measured by the mean frequencies of the pre-/post-EEGs.

Subject #1, because of a skull defect which allows high frequency activity to be observed without normally occurring attenuation, showed

Table 2. Chi Square, Number of Counts, Percent Time Values and Mean Frequencies for Subject 1, Channel 1 (C_3-C_4), Pre-/Posttraining.

Frequency Bands	Pre			Post			Pre-Post Change
	%	Counts	χ^2	%	Counts	χ^2	
1.0 - 3.012 Hz	0.8	98	43.17**	.23	205	59.23***	+
3.012 - 6.024 Hz	.29	705	9.19	.30	617	12.62*	-
6.024 - 9.091 Hz	.28	1128	20.2**	.20	677	13.84*	-†
9.091 - 12.195 Hz	.16	925	4.51	.12	592	6.75	-
12.195 - 15.152 Hz	.10	756	10.9	.08	481	14.91*	-
15.152 - 18.182 Hz	.05	479	11.14*	.03	242	15.28**	-
18.182 - 20.0 Hz	.03	349	22.12***	.05	427	30.61***	+†

Mean Frequencies	
Pre	Post
10.5	10.3

Overall Chi Square Pre/Post

$\chi^2 = 264.36$

df = 35

$p \leq .001$

*p ≤ .05

**p ≤ .01

***p ≤ .001

† = hypothesized direction

Table 3. Chi Square, Number of Counts, Percent Time Values and Mean Frequencies for Subject 1, Channel 2 (T_3-T_4), Pre-/Posttraining.

Frequency Bands	Pre			Post			Pre-Post Change
	%	Counts	χ^2	%	Counts	χ^2	
1.0 - 3.012 Hz	.07	91	80.93***	.28	264	109.62***	+
3.012 - 6.024 Hz	.30	788	30.41***	.33	756	41.69***	-†
6.024 - 9.091 Hz	.31	1262	14.56*	.21	770	20.99***	-†
9.091 - 12.195 Hz	.16	931	16.33**	.09	489	23.46***	-
12.195 - 15.152 Hz	.09	681	22.28***	.05	370	32.11***	-
15.152 - 18.182 Hz	.04	387	8.58	.02	175	12.36*	-
18.182 - 20.0 Hz	.03	284	2.92	.03	246	4.22	-

Mean Frequencies

Pre	Post
10.0	9.0

Overall Chi Square Pre/Post

$\chi^2 = 420.13$

df = 35

$p \leq .001$

*p ≤ .05

**p ≤ .01

***p ≤ .001

† = hypothesized direction

Table 4. Chi Square, Number of Counts, Percent Time Values and Mean Frequencies for Subject 2, Channel 1 (C_3-C_4), Pre-/Posttraining.

Frequency Bands	Pre			Post			Pre-Post Change
	%	Counts	χ^2	%	Counts	χ^2	
1.0 - 3.012 Hz	.04	48	17.63**	.08	108	19.81**	+
3.012 - 6.024 Hz	.20	540	25.09***	.28	705	28.14***	+
6.024 - 9.091 Hz	.35	1549	29.68***	.32	1344	33.26***	-†
9.091 - 12.195 Hz	.24	1390	37.85***	.16	942	46.35***	-
12.195 - 15.152 Hz	.09	682	18.7**	.08	654	20.93**	-
15.152 - 18.182 Hz	.05	457	6.08	.04	340	6.82	-
18.182 - 20.0 Hz	.03	342	3.94	.04	378	4.42	+†

Mean Frequencies

Pre	Post
10.5	10.2

Overall Chi Square Pre/Post

$\chi^2 = 298.73$

df = 35

$p \leq .001$

*p ≤ .05

**p ≤ .01

***p ≤ .001

† = hypothesized direction

Table 5. Chi Square, Number of Counts, Percent Time Values and Mean Frequencies for Subject 2, Channel 2 (T_3-T_4), Pre-Posttraining.

Frequency Bands	Pre			Post			Pre-Post Change
	%	Counts	χ^2	%	Counts	χ^2	
1.0 - 3.012 Hz	.03	37	44.5***	.12	154	53.7***	+
3.012 - 6.024 Hz	.22	559	52.24***	.32	790	62.58***	+
6.024 - 9.091 Hz	.33	1354	11.12*	.28	1127	13.41*	-†
9.091 - 12.195 Hz	.20	1131	12.3*	.13	754	14.83*	-
12.195 - 15.152 Hz	.12	884	9.97	.08	564	11.59*	-
15.152 - 18.182 Hz	.07	627	9.58	.05	423	11.49*	-
18.182 - 20.0 Hz	.04	389	.856	.03	321	1.03	-

Mean Frequencies	
Pre	Post
11.0	10.0

Overall Chi Square Pre/Post

$\chi^2 = 311.11$

df = 35

$p \leq .001$

*p ≤ .05

**p ≤ .01

***p ≤ .001

† = hypothesized direction

Table 6. Chi Square, Number of Counts, Percent Time Values and Mean Frequencies for Subject 4, Channel 1 (C_3-C_4), Pre-Posttraining.

Frequency Bands	Pre			Post			Pre-Post Change
	%	Counts	χ^2	%	Counts	χ^2	
1.0 - 3.012 Hz	.10	206	49.84***	.05	57	46.23***	-†
3.012 - 6.024 Hz	.28	901	30.26***	.25	658	28.07***	-†
6.024 - 9.091 Hz	.23	1056	11.26	.22	923	10.44	-†
9.091 - 12.195 Hz	.22	920	53.03***	.26	1509	49.21***	+†
12.195 - 15.152 Hz	.08	630	22.36***	.13	950	20.75***	+†
15.152 - 18.182 Hz	.05	530	1.16	.06	552	1.72	+†
18.182 - 20.0 Hz	.04	357	4.15	.03	309	3.85	-

Mean Frequencies

Pre	Post
9.88	10.9

Overall Chi Square Pre/Post

χ^2 = 330.32

df = 35

$p \leq .001$

*p ≤ .05

**p ≤ .01

***p ≤ .001

† = hypothesized direction

Table 7. Chi Square, Number of Counts, Percent Time Values and Mean Frequencies for Subject 4, Channel 2 (T_3-T_4), Pre-/Posttraining.

Frequency Bands	Pre			Post			Pre-Post Change
	%	Counts	χ^2	%	Counts	χ^2	
1.0 - 3.012 Hz	.15	200	23.97***	.08	102	21.19***	-†
3.012 - 6.024 Hz	.34	945	29.32***	.31	744	25.92***	-†
6.024 - 9.091 Hz	.20	823	1.52	.24	1008	1.34	+
9.091 - 12.195 Hz	.14	800	21.99***	.21	1212	19.45**	+†
12.195 - 15.152 Hz	.11	550	7.48	.10	767	6.61	+†
15.152 - 18.182 Hz	.04	404	1.61	.05	424	1.54	+†
18.182 - 20.0 Hz	.02	260	1.68	.02	247	1.81	-

Mean Frequencies	
Pre	Post
9.60	10.2

Overall Chi Square Pre/Post

$\chi^2 = 167.43$

df = 35

$p \leq .001$

*p ≤ .05

**p ≤ .01

***p ≤ .001

† = hypothesized direction

Table 8. Chi Square, Number of Counts, Percent Time Values and Mean Frequencies for Subject 5, Channel 1 (C_3-C_4), Pre-/Posttraining.

Frequency Bands	Pre			Post			Pre-Post Change
	%	Counts	χ^2	%	Counts	χ^2	
1.0 - 3.012 Hz	.17	224	53.56***	.06	76	45.65***	-†
3.012 - 6.024 Hz	.38	968	54.18***	.31	779	46.19***	-†
6.024 - 9.091 Hz	.25	1064	9.8	.34	1364	8.373	+
9.091 - 12.195 Hz	.11	652	6.06	.15	863	5.2	+†
12.195 - 15.152 Hz	.04	348	8.95	.07	519	8.47	+†
15.152 - 18.182 Hz	.03	272	6.16	.04	366	5.45	+†
18.182 - 20.0 Hz	.02	198	22.49***	.04	400	20.53***	+†

Mean Frequencies

Pre	Post
8.8	10.0

Overall Chi Square Pre/Post

$\chi^2 = 301.35$

df = 35

$p \leq .001$

*p ≤ .05

**p ≤ .01

***p ≤ .001

† = hypothesized direction

Table 9. Chi Square, Number of Counts, Percent Time Values and Mean Frequencies for Subject 5, Channel 2 (T_3-T_4), Pre-/Posttraining.

Frequency Bands	Pre			Post			Pre-Post Change
	%	Counts	χ^2	%	Counts	χ^2	
1.0 - 3.012 Hz	.10	142	20.83***	.05	57	23.09***	-†
3.012 - 6.024 Hz	.37	936	45.23***	.26	656	43.70***	-†
6.024 - 9.091 Hz	.26	1098	5.84	.32	1258	4.86	+
9.091 - 12.195 Hz	.12	743	10.20	.16	915	6.62	+†
12.195 - 15.152 Hz	.07	504	25.91***	.10	708	16.8**	+†
15.152 - 18.182 Hz	.05	441	8.29	.06	556	7.76	+†
18.182 - 20.0 Hz	.03	348	11.49***	.05	530	10.35**	+†

Mean Frequencies

Pre	Post
9.9	11.0

Overall Chi Square Pre/Post

$\chi^2 = 244.50$

df = 35

$p \leq .001$

*p ≤ .05

**p ≤ .01

***P ≤ .001

† = hypothesized direction

Table 10. Chi Square, Number of Counts, Percent Time Values and Mean Frequencies for Subject 6, Channel 1 (C_3-C_4), Pre-/Posttraining.

Frequency Bands	Pre			Post			Pre-Post Change
	%	Counts	χ^2	%	Counts	χ^2	
1.0 - 3.012 Hz	.20	260	90.35***	.05	67	74.42***	-†
3.012 - 6.024 Hz	.36	924	24.48***	.32	828	20.19**	-†
6.024 - 9.091 Hz	.28	1220	11.03	.40	1698	9.34	+
9.091 - 12.195 Hz	.10	603	4.37	.14	803	3.58	+†
12.195 - 15.152 Hz	.03	271	13.47*	.06	464	11.12*	+†
15.152 - 18.182 Hz	.02	155	9.82	.03	275	9.09	+†
18.182 - 20.0 Hz	.01	92	1.33	.01	136	1.11	+†

Mean Frequencies	
Pre	Post
8.0	9.0

Overall Chi Square Pre/Post

$\chi^2 = 283.143$

df = 35

$p \leq .001$

*p ≤ .05

**p ≤ .01

***p ≤ .001

† = hypothesized direction

Table 11. Chi Square, Number of Counts, Percent Time Values and Mean Frequencies for Subject 6, Channel 2 (T_3-T_4), Pre-/Posttraining.

Frequency Bands	Pre			Post			Pre-Post Change
	%	Counts	χ^2	%	Counts	χ^2	
1.0 - 3.012 Hz	.11	138	46.64***	.02	31	40.03***	-†
3.012 - 6.024 Hz	.32	829	55.02***	.22	574	47.31***	-†
6.024 - 9.091 Hz	.28	1153	10.57	.36	1502	7.88	+
9.091 - 12.195 Hz	.14	810	5.91	.19	1095	5.08	+†
12.195 - 15.152 Hz	.09	652	2.40	.11	838	1.41	+†
15.152 - 18.182 Hz	.04	411	5.88	.06	588	5.06	+†
18.182 - 20.0 Hz	.02	247	1.6	.03	330	1.38	+†

Mean Frequencies

Pre	Post
9.9	10.7

Overall Chi Square Pre/Post

$\chi^2 = 243.85$

df = 35

$p \leq .001$

*p ≤ .05

**p ≤ .01

***p ≤ .001

† = hypothesized direction

a significant reduction in average frequency. For this subject this shift was considered a normalizing one. He, unlike the other subjects on the initial EEG, had not manifested overall slowing but instead produced an increased amount of faster activity in the 7-13 Hz range.

Subject #2 displayed a slowing of activity with pre-/post-measurement, and this result does not appear to support the hypothesis. Dividing the overall frequency pattern into approximately three Hertz bands allowed a finer discrimination to see where shifts did or did not take place.

Subjects #4, #5, and #6 displayed a significant diminution of low frequency responses in the frequency bands 1.0-3.01 Hz and 3.01-6.02 Hz Subjects #1 and #2 did not exhibit the predicted change in these bands, instead their post measures showed an augmentation of slow frequency responses. Both subjects #1 and #2 did, however, evidence a significant diminution of response in the 6.02-9.10 Hz range.

Subject #4 was the only person who displayed a significant increase in the number of counts in the 9.10-12.20 Hz range. Subjects #5 and #6 produced a positive but nonsignificant count increase in this band. Subjects #1 and #2 exhibited a significant decrease in the number of counts, contrary to hypothesis 1.

Other Associated Changes in EEG Pattern Suggested by Frequency Analysis

Though not reinforced, a significant augmentation in the number of counts was demonstrated within the 12.2-15.2 Hz and 18.2-20 Hz range for subject #5 on both channels. Subjects #4 and #6 demonstrated a significant increase in the 12.20-15.15 Hz range in channel 1. Though nonsignificant, subjects #4, #5, and #6 produced a consistent trend

toward the faster frequencies from 9.10 Hz on. Subjects #1 and #2 demonstrated the opposite effect; subject #1 evidenced a significant diminution on channel 2 in the ranges of 9.10-18.18 Hz and on channel 1 in the 12.20-20.0 Hz range. Subject #2 displayed a significant slowing in the 9.10-12.20 Hz range and a nonsignificant slowing trend in the 12.20-18.18 Hz range.

Since the Chi-square changes represented both distributional and level changes, the mean frequency shift was more instructive in describing those changes. It did, however, reflect the combined effects of all frequency bands and, in the specific case of alpha diminution, may have distorted the picture.

Unfortunately, in dealing with dependent data, percentages were employed and a shift in one frequency band was expressed as a change in another. If the EEG is perceived on a continuum from hypersynchrony (many synaptic potentials occuring together) to synchrony (with synaptic potentials largely out of phase), attempts to shift toward a midfrequency range may result in diminished desynchronization.

Data with Respect to Hypothesis 2

Hypothesis 2 made the following predictions: 1) overall, seizure incidence will diminish over time and be significantly less at the completion of the study, and 2) this decrease will be a function of the training condition (see Table 12 and Appendix B) and the abundance of alpha and theta activity throughout all conditions (see Table 13).

Overall Seizure Incidence

Table 12 illustrates the test of the hypothesis that no shift in the seizure rate occurs throughout the study. That is, the null

Table 12. Chi-square Measures of Seizure Occurrence by Days and Conditions

	Condition	Seizures per Condition	Days in Condition	Expected	$\frac{(O-E)^2}{E}$	Average Seizure/ Day	
(01)							
Baseline	A	59	31	54.60	0.35	1.84	
Treatment	B_1	60	31	54.60	0.53	1.93	
Noncontingent	C	13	8	14.09	0.084	1.62	
Treatment	B_2	15	12	21.13	1.77	1.25	
Theta Suppression	D	18	6	10.56	5.24*	3.00	
	TOTAL	155	88		$\chi^2 = 7.97$		N.S.
					df = 4		
(02)							
Baseline	A	27	41	21.94	1.16	.65	
Treatment	B_1	17	31	16.59	.16	.54	
Noncontingent	C	1	6	3.21	4.88*	.16	
Treatment	B_2	6	14	7.49	2.22	.42	
Theta Suppression	D	2	7	3.74	3.02	.28	
	TOTAL	53	99		$\chi^2 = 11.44$		$p \leq .025$
					df = 4		
(04)							
Baseline	A	207	31	80.59	198.28***	6.67	
Treatment	B_1	27	44	114.39	66.76***	.061	
Noncontingent	C	0	7	18.19	18.19***	0	
Treatment	B_2	0	8	20.79	20.75***	0	
	TOTAL	234	90		$\chi^2 = 304.02$		$p \leq .005$
					df = 3		

Table 12. --Continued.

	Condition	Seizures per Condition	Days in Condition	Expected	$\frac{(O-E)^2}{E}$	Average Seizure/ Day	
(05)							
Baseline	A	19	31	9.98	8.14*	.61	
Treatment	B_1	8	37	11.92	1.29	.21	
Noncontingent	C	1	8	2.57	.96	.125	
Treatment	B_2	1	14	4.51	2.73	.07	
	TOTAL	29	90		$\chi^2 = 11.77$		$p \leq .01$
					df = 3		
(06)							
Baseline	A	147	31	110.91	11.74**	4.74	
Treatment	B_1	106	31	110.91	.21	3.41	
Noncontingent	C	16	8	28.62	5.56*	2.00	
Treatment	B_2	38	12	42.93	24.30***	3.16	
Theta Suppression	D	15	8	28.62	6.48*	1.87	
	TOTAL	322	90		$\chi^2 = 48.29$		$p \leq .005$
					df = 4		

* $p \leq .05$
** $p \leq .025$
*** $p \leq .005$

Table 13. Correlations Between EEG Variables and Seizure Frequency

Variables	All Subjects	1	2	4	5	6
Alpha percentage	-0.30****	-0.02	-0.26	-0.59***	-0.12	-0.37*
Average frequency	0.31****	0.007	-0.09	0.55***	-0.07	0.27
Average amplitude	0.09	-0.11	0.36*	0.45***	-0.07	0.36*
Theta percentage	-0.03	-0.18	0.42**	-0.07	0.02	-0.08
Theta count	-0.01	-0.18	0.34*	-0.08	0.09	0.03
Number of observations	177	35	35	32	31	36

*p ≤ .05
**p ≤ .01
***p ≤ .001
****p ≤ .0001

hypothesis that the treatment administered in a condition had no effect upon seizure rate. The expected value reported for each condition in the table then is the product of the total number of seizures each subject had throughout the study multiplied by the percentage of total time occupied by each condition. This test appears conservative since the influence of the experimental conditions was of interest rather than a pre-post test of seizure rate. Appendix B presents a graphic summary of the observed and expected Chi-square values from Table 12 for each subject.

Subject #1 produced a nonsignificant Chi-square indicating no change in seizure incidence over the course of treatment. Subjects #2, #4, #5, and #6 all displayed significant Chi-square values indicating a change in the frequency of seizures over the time course of training. Taking into account the average seizures per day, subjects #2, #4, #5, and #6 displayed an overall decrease. Subject #1, in effect, displayed no change in the average seizures per day.

Seizure Incidence as a Function of Training Conditions

During baseline condition A, subjects #4, #5, and #6 produced a significant departure from expectation by exhibiting a greater number of reported seizures as measured by the average seizure per day figures. This result was in keeping with the predictions of hypothesis 2 since the baseline period (A) was assumed to measure the frequency of seizures prior to intervention. Subjects #1 and #2 evidenced nonsignificant results during this condition, which indicated no elevation in seizure activity during the baseline period.

During the first treatment condition (B_1), subject #4 demonstrated a highly significant departure from expectation in seizure frequency.

This result was consistent with the predictions of hypothesis 2. Subjects #2, #5, and #6 produced a nonsignificant diminution in seizure incidence during treatment B_1. Subject #1 displayed an increase in seizure production during this condition, a result which was contradictory to the expectations of hypothesis 2.

During condition C, the noncontingent feedback phase, subjects #2, #4, and #6 displayed significant decreases in seizure rate--a result not predicted during this condition. Subjects #1 and #5 displayed nonsignificant decreases in seizure frequency--a result that was also not predicted by the noncontingent feedback of condition C. This matter will be explored further in the next chapter.

During the second feedback condition (B_2) only subjects #4 and #6 evidenced a significant negative correlation. For these subjects these results indicate a decrease in seizure frequency.

Of the three subjects taking part in condition D, the direct suppression of theta activity without concurrent alpha enhancement, subject #6 was the only one to display a significant diminution in seizure incidence. Subject #1 produced a significant increase in seizure frequency. From the standpoint of hypothesis 2, only subject #6's result was predicted.

Table 13 presents the correlations of EEG variables with seizure frequency over all subjects and each individual subject. For all subjects there was a significant negative correlation between alpha percentage and seizure incidence. As alpha percent diminished, the seizure rate increased. On an individual basis, subjects #4 and #6 evidenced significant negative correlations while subjects #1, #2, and #5 produced negative but not significant ones.

For all subjects there was a significant positive correlation between average frequency and seizure incidence. In effect, as the average frequency increased, the rate of seizures also increased. On an individual basis this result was particularly displayed by subject #4.

Though the overall correlation between average amplitude and seizure frequency was not significant, subjects #2, #4, and #6 all displayed significant positive correlations. This indicated that as the average amplitude in their EEG waves increased there was a similar increase in seizure frequency.

In reference to theta percentage and theta count subject #2 displayed a significant positive correlation indicating that there was an increase in seizure rate as theta production increased. This was the only subject for which this was true.

Data with Respect to Hypotheses 3 and 4

Hypotheses 3 and 4 were evaluated in two ways: one, the significance of the slopes within each condition was determined. These data allowed statements to be made as to the rate of change within each condition testing it against the null hypothesis that slope = 0. Second, the significance of the difference in slopes between conditions and the difference in means was determined. These data, too, allowed statements to be made as to the possible differences in treatment versus baseline conditions testing against the null hypothesis that the differences between slopes and mean = 0 (Kazdin, 1976). The slope data obtained within treatment conditions was presented first, followed by the combined presentation of the contrasts between the slopes and means for baseline versus all treatment conditions.

Data with Respect to Baseline Condition A

Condition A stated that during the initial three-trial baseline, subjects will display a zero slope (see Appendix C within condition for data). Subjects #1 and #5 produced the predicted nonsignificant slopes indicating no change in alpha percent during the baseline period. Subjects #2, #4, and #6 displayed significant slopes with subject #2 producing a positive one, which indicated an increase in alpha and subjects #4 and #6 producing negative slopes which indicated a diminution in alpha percent.

Data with Respect to Condition B_1

During the eighteen-trial treatment condition, subjects will display a positive slope and an increase in level change from baseline condition A. In this context, level and mean represented the same concept--namely, that a change occurs in the mean or average level of a given dependent variable between one condition and other. It should be noted that this definition differs from the one usually applied in the classical time-series literature, which refers to a level change as occurring when a discontinuity exists between the data at the point when a treatment is introduced (Hersen and Barlow, 1976).

This condition consisted of a five-minute segment pre-/post-alpha feedback where no feedback was forthcoming. These segments were termed pretreatment B_1 and posttreatment B_1, respectively. Between these no-feedback segments there was a forty-minute alpha enhancement condition to be called treatment B_1.

During the pretreatment B_1 period for within condition slopes, subject #5 displayed no change in the rate of alpha production, a

result that was not coincident with a combined feedback/no-feedback condition as one might have expected, as one might have expected a gradual increase in alpha acquisition over time. Subjects #1, #2, #4, and #6 all displayed a significant increase of alpha percent, a positive slope. This result was coincident with the hypothesis.

During the treatment B_1 condition, subjects #1, #4, #5, and #6 displayed the predicted enhancement of alpha percent during this feedback phase. Subject #2 yielded a nonsignificant slope indicating no change in alpha percent during this phase.

During the posttreatment B_1 condition, subjects #1, #2, and #6 displayed nonsignificant slopes indicating no change in alpha production. As with the expectations of pretreatment B_1, this result was not in keeping with the hypothesis. Subjects #4 and #5 produced a significant positive slope indicating an increase in alpha percent. Because these subjects also produced a significant treatment B_1 effect, this carryover to an immediate posttreatment no-feedback phase may well have been a function of learning and, therefore, consistent with the predictions of hypothesis 3.

Data with Respect to Condition C

During the four-trial noncontingent condition, subjects will produce a negative slope as compared with Condition B. As with the treatment B_1 condition, the noncontingent phase was divided into three parts: 1) pre-noncontingent phase of five minutes with no feedback; 2) forty-minute noncontingent phase, when sham feedback was given; and 3) post-noncontingent phase of five minutes when no feedback was forthcoming.

Subject #1 yielded a significant negative slope, indicating a diminution in alpha percentage during this phase. Given this subject's prior history of a skull defect and inability to filter out faster waves, this result might have been expected. During this time this subject began again to produce fast activity very similar to that produced during condition A. Subjects #2, #4, #5, and #6 yielded nonsignificant slopes.

During the noncontingent feedback phase, subjects #1 and #5 produced significant negative slopes indicating a diminution in alpha production as a result of this false feedback stage. This result was in keeping with the tenets of hypothesis 3. Subjects #2 and #4 yielded nonsignificant slopes, indicating no change in alpha production as compared with baseline measures. Subject #6 displayed a significant positive slope which indicated an increase in alpha percent.

During the post-noncontingent phase, subjects #1, #2, #5, and #6 all produced nonsignificant slopes indicating no change in alpha wave percentage. These results were predicted especially in the case of subject #5, who produced a significant diminution in alpha in the prior phase and a nonsignificant slope in the pre-noncontingent phase. These results, taken together, indicated that for this subject the use of noncontingent feedback had a direct effect upon his response. When he was released from this feedback, he reverted to a no change status. Subject #4 produced a significant negative slope indicating a continued diminution of alpha percent during this latter phase.

Data with Respect to Condition B_2

During the six-trial contingent alpha enhancement condition subjects will display a positive slope and a significant increase in level from baseline condition A.

During the pretreatment phase subjects #1, #2, #4, #5, and #6 all yielded nonsignificant slopes indicating no change in alpha production.

During the contingent feedback phase, subjects #1, #2, and #4 yielded nonsignificant slopes indicating no change in alpha production as a result of feedback. Subjects #5 and #6 produced significant negative slopes demonstrating a decrease in alpha wave production. These results are contrary to the direction predicted by the contingent phase of hypothesis 3.

During the posttreatment phase all subjects yielded nonsignificant slopes, indicating no change in alpha production during this phase.

Data with Respect to Hypothesis 4

Data with Respect to Condition A

During the initial three-trial baseline, subjects' data level will be stable and a least-squares line fitted to the data will have a zero slope.

Subjects #2, #4, and #6 displayed the predicted nonsignificant slopes indicating no change in theta percent during the baseline period. Subject #1 produced a significant positive slope indicating an increasing amount of theta production in the absence of feedback. Subject #5 exhibited a significant negative slope indicating a decreased theta production during the baseline period.

Data with Respect to Condition B_1

During the eighteen-trial treatment condition, subjects will display a negative slope and a decrease in level from baseline condition A.

This was a tripartate condition consisting of a five-minute segment pre-/posttheta suppression where no feedback was forthcoming. These segments will be termed pretreatment B_1 and posttreatment B_1, respectively. Between these no-feedback segments there was a forty-minute theta suppression condition to be called treatment B_1.

During the pretreatment B_1 phase subjects #1, #2, #4, #5, and #6 all displayed nonsignificant slopes. This result was not in keeping with hypothesis 4. Because of the contiguity in time of treatment B_1, a gradual increase in slope indicating a learned generalization was to have been expected.

During the treatment B_1 phase, subjects #1 and #4 displayed a significant diminution in theta production, a result which is coincident with hypothesis 4. Subjects #2, #5, and #6 evidenced nonsignificant slopes indicating no change in the amount of theta produced.

During posttreatment B_1, all subjects displayed nonsignificant slopes indicating no change in theta production during this no-feedback phase, again inconsistent with the hypothesis.

Data with Respect to Condition C

During this four-trial noncontingent phase, subjects will produce a positive slope and a decrease in level from baseline condition A.

Like the treatment B_1 condition, the noncontingent phase was divided into three parts: 1) pre-noncontingent phase of five minutes

with no feedback; 2) forty-minute noncontingent phase where feedback was supplied; and 3) post-noncontingent phase of five minutes with no feedback.

During the pre-noncontingent phase all subjects produced non-significant slopes indicating no change in theta production, a result coincident with hypothesis 4.

During the noncontingent feedback phase, subject #5 displayed a significant negative slope indicating a diminution in the amount of theta produced. Subjects #1, #2, #4, and #6 evidenced nonsignificant slopes indicating no change due to noncontingent feedback.

During the post-noncontingent phase, all subjects displayed nonsignificant slopes indicating no change in the production of theta.

Data with Respect to Condition B_2

During the six-trial contingent phase, subjects will display a negative slope and a significant decrease in level from baseline condition A.

As in the B_1 condition, B_2 was contingent and divided into three phases: 1) a five-minute pretreatment phase with no feedback; 2) forty-minute treatment phase with theta suppression; and 3) a five-minute posttreatment phase with no feedback.

During the pretreatment B_2 phase, subject #1 produced a significant diminution in theta percent, and subject #5 displayed a positive slope which indicated an increase in theta production. Subjects #2, #5, and #6 produced nonsignificant slopes which indicated no change in the production of theta.

During the treatment phase of B_2, subjects #1 and #5 displayed significant negative slopes indicating a diminution in theta production during this phase. This result was consistent with the tenets of hypothesis 4. Subjects #2, #4, and #6 displayed nonsignificant slopes indicating no change in theta production due to the combined alpha augmentation and theta suppression.

During the posttreatment phase of condition B_2, subjects #2 and #5 produced negative slopes indicating a decrease in theta production in this phase. Subjects #1, #4, and #6 displayed nonsignificant slopes showing no change in theta production.

Data with Respect to Condition D

During the three- to four-trial theta suppression condition, subjects will display a negative slope and a significant decrease in level from condition A.

During the pretreatment phase of condition D, subjects #1, #2, and #6, the only ones taking part in this condition, all displayed nonsignificant slopes indicating no change during this phase.

During the treatment phase of condition D, subjects #1 and #6 both evidenced a significant negative slope indicating a diminution in the production of theta. This result was consistent with hypothesis 4. Subject #2 produced a nonsignificant slope showing no suppression in theta production.

During the posttreatment phase of condition D, all subjects displayed nonsignificant slopes indicating no change in theta during this phase.

Between Slope and Mean Differences

Though all pair-wise comparisons were determined, only the differences between conditions A-B_1, A-C, A-B_2 and A-D, that is, the differences in baseline and treatment conditions, were reported for alpha percent and theta percent. Appendix D presented the selected pair-wise contrasts between these conditions for slopes and levels.

Data with Respect to A-B_1 Alpha/Theta Comparisons

The pretreatment phase condition B_1 yielded a significant accelerated alpha slope for subjects #1, #4, and #6. Subject #2 displayed a deceleration in alpha production in this comparison.

During the treatment phase subjects #1, #4, #5, and #6 evidenced a significant accelerated alpha response slope as compared to baseline. Subject #2 yielded a significant deceleration in slope in this comparison.

During the posttreatment B_1 phase subjects #1, #4, and #5 yielded slopes that were accelerating significantly when compared to baseline recordings.

In reference to the slope of theta production during the pretreatment B_1 phase, subject #1 produced a significant decelerated slope which indicated that this phase had an effect on changing responding in a negative direction. Subjects #2 and #5 displayed an A-B_1 slope comparison which indicated a greater acceleration of theta as compared to baseline.

During the treatment phase the slope of theta production was significantly decelerated for subjects #1, #4, and #6. Subjects #2

and #5 displayed significantly accelerating slopes which indicated an increase in theta production as compared to baseline.

The posttreatment A-B_1 comparison yielded a significant deceleration in slope for subject #6. Subject #5 produced an accelerated slope which indicated an increase in theta responding.

Data with Respect to A-C Alpha/Theta Comparisons

The pre-noncontingent phase yielded a significant deceleration in slope for alpha percent for subjects #1 and #2. Subjects #4 and #6 displayed an acceleration in alpha production as compared to baseline.

The noncontingent phase of condition C yielded a significant deceleration in the slope of alpha percent for subjects #1 and #2 as compared to baseline. Subject #6 displayed significantly accelerating slopes which indicated a continued increase in alpha production.

The post-noncontingent phase yielded a significant deceleration in the slope of alpha for subject #2. Subject #6 displayed an accelerated slope as compared to baseline.

The A-C slope comparisons of theta production during pre-noncontingent phase of condition C yielded a significant deceleration for subject #1 as compared to baseline. Subject #6 displayed an accelerated slope which indicated greater theta responding as compared to baseline. Subject #5 displayed no significance difference in slopes but did evidence a significant level difference, indicating greater theta responding though no change in direction of response.

During the noncontingent feedback phase subject #1 displayed a significant deceleration in slope which indicated a decrease in theta

productivity as compared to baseline. Subject #5 displayed a lessened deceleration as compared to baseline. Subject #2 produced an accelerated comparison which indicated an increase in theta responding.

During the post-noncontingent phase subject #1 displayed a difference in slopes that indicated a deceleration of theta production. Subjects #5 and #6 produced a significant acceleration in theta production as compared to baseline.

Data with Respect to A-B_2 Alpha/Theta Comparisons

The pretreatment phase of condition B_2 yielded a significant acceleration in the slope of alpha production for subjects #4, #5, and #6. Subject #2 yielded a slope comparison which indicated a deceleration in alpha responding as compared to baseline.

During the treatment phase of condition B_2 subjects #4, #5, and #6 yielded significant accelerating slopes as compared to baseline. Subject #2 displayed a deceleration which indicated a lessening of alpha production in this comparison.

The posttreatment B_2 comparison yielded a significant acceleration in slope for subjects #4 and #6. Subjects #2 and #5 displayed a comparative deceleration in alpha production.

In reference to the A-B_2 slope comparisons of theta production during the pretreatment phase, subject #1 displayed a significant deceleration in slope as compared to baseline. Subjects #2, #4, and #5 produced A-B_2 slope comparisons that indicated an acceleration in theta production in the B_2 pretreatment phase.

During the treatment phase subjects #1, #4, #5, and #6 displayed significant decelerations in slope indicating a lessened production of theta. Subject #2 displayed a significant slope difference which indicated an acceleration in theta percent in the comparison.

The posttreatment A-B_2 comparison yielded a significant deceleration in responding for subjects #1 and #6. Subject #5 displayed a difference in slopes that indicated an acceleration in responding during the posttreatment B_2 condition.

Data with Respect to A-D Alpha/Theta Comparisons

Since only three subjects (#1, #2, and #6) took part in the D theta suppression condition, A-D comparisons were confined to them.

Subjects #1 and #2 displayed a significant deceleration in the slope of alpha percent during the pretreatment phase. Subject #6 yielded a slope comparison which indicated a significant acceleration in this phase as compared to baseline.

During the treatment phase subjects #1 and #2 again yielded a significant deceleration in alpha production as compared to baseline. Subject #6 again yielded a significant acceleration in alpha production.

The posttreatment phase yielded a significant deceleration in the slope of alpha percent for subjects #1 and #2. Subject #6 displayed an acceleration of slope which indicated an increase in alpha responding as compared to baseline.

During the pretreatment phase of condition D the A-D comparison in slopes for theta production yielded a significant deceleration in slope for D condition as compared to baseline for subject #1.

Subject #2 yielded a t-ratio of slope comparisons that indicated that there was an acceleration of theta production.

During the treatment phase the A-D slope comparison yielded significant deceleration in responding for subjects #1, #2, and #6 which indicated a lessened production of theta during this phase.

The posttreatment phase of the A-D slope comparisons yielded a significant t-ratio which indicated a significant deceleration in the slope of theta for subject #1. The remaining two subjects yielded insignificant differences.

Of the fifty-four individual pair-wise contrasts for alpha percent, none of the mean differences between slopes were significant. This lack of difference, combined with the fact that the vast majority of the differences between slopes were significant, harkened to a change in direction of the individual slopes but not to level. This result may well have been due to a baseline period that did not allow differences in baseline versus treatment conditions to be discerned.

Of the fifty-four individual pair-wise contrasts for theta percent, only three (subject #5 A-C, significant increase in slope and level; subject #5 A-B_2, significant increase in slope and level; and subject #2 A-B_2 significant increase in theta percent) proved to be significant. As with measurement of alpha percent, the inadequacy of baseline data may well have accounted for this result.

If physiological feedback can be considered a slowly learned discrimination task, the results of significant slopes and nonsignifican level might have been expected. A skill that required time to develop would have produced stable maximal levels before the cumulative effect of training produced significant differences in the means of

conditions. The fact that there were many significant within condition slope changes was seen as supporting this contention.

CHAPTER IV
DISCUSSION

The present study provides additional evidence for the efficacy of EEG physiological feedback training in the midfrequency range as a means of controlling seizures. To explore this matter further, this chapter will be divided into three sections: first, a discussion of individual hypotheses and the basis for their support or rejection; second, critique of the experimental design and suggestions for improvement; lastly, suggestions for future research.

Discrepancies Between Hypotheses and Results

Hypothesis #1 predicted an increase in EEG frequency during feedback training. The overall results support this contention in three of five subjects (#4, #5, #6). These subjects increased their average frequencies in this pre-/postmeasurement condition.

Subject #1 displayed a decrease in average EEG frequency across both bipolar electrode sites (T_3-T_4, C_3-C_4) in the pre-/postfrequency analysis. As stated before, this subject's skull defect caused him not to attenuate higher frequency activity, and, hence, the pretraining EEG displayed this overabundance of fast-wave activity. The alpha band (8-13 Hz) is essentially the middle range of the typical frequencies recorded in the human EEG; an individual with predominantly desynchronized beta activity when presented with the contingencies of alpha feedback is likely to decrease his frequency to maximize

feedback in accordance with established set. The question that is of course left unanswered in this subject's case is whether diminishing the frequency reduces the subject's seizure rate. Since no significant decrease in seizure rate occured, it appears his response to the task requirements was not beneficial.

Subject #2 was unable to increase mean frequency pre/post. The conclusion one draws from this and the fact that he was also virtually unable to control alpha and theta training contingencies is that the procedure, as it is presently constructed, does not work for him. This subject had a successful diminution of seizure rate only in the C or noncontingent condition. Heightened arousal level resulting from stimulus novelty (orienting reflex) cannot account for this result in the subject's response because his average frequency decreased during noncontingent periods.

Seizure Incidence and Treatment Condition Analysis

The finding that seizure rate was positively correlated with average frequency is a potentially important result. This is in contrast to Sterman's most recent statement (1978) where he indicates an increase in average frequency is negatively correlated with seizure rate. In discussing this discrepancy it is most important to note the differences in the subject's seizure types chosen for the two studies. Sterman chose subjects heterogeneous in seizure type ranging from the centrencephalic to focal type. The present study chose subjects only with focal seizures because it was thought best to test the role of physiology feedback on a homogeneous population. It is clear that the mechanism of seizure propagation is quite different

in centrencephalic versus focal seizures (Jasper, 1969). To attempt to make any generalizations as to the efficacy of any one approach, a distinctly homogeneous population is a prerequisite.

From the standpoint of focal seizure patients, the results indicated an increase in seizure rate with an increase in average frequency. As the variability of alpha production decreased, the seizure rate also increased. From the standpoint of the malleability of the control mechanisms of the brain, this result supports the notion that a lessening of synchronous activity reduces the ability to inhibit seizures for these subjects (Jasper, 1969). In effect, as the thalamocortical synchronization reflected in alpha percentage decreases during the production of faster, essentially desynchronous activity, seizure rate increases. This result argues for a midrange hypothesis, in effect a normalization, for individuals with focal motor seizures.

Alpha Acquisition

Alpha percentage was significantly and positively correlated with feedback in the B_1 treatment condition for four out of five subjects. These results indicated an ability to acquire the alpha response as a function of the treatment condition. When noncontingent reinforcement was instituted, there was a significant within condition shift and a between condition slope difference for three of these four subjects. This same overall result was not forthcoming for the second contingent condition (B_2) and argues to the brevity of this condition as a cause for its failure. This matter will be explored further in the section on design critique.

Subject #2 displayed no effect from alpha acquisition training during any of the treatment conditions. There were no readily explainable reasons for this result; this subject assiduously maintained appointments, understood the task requirements and very much wanted improvement. As will be stated later, the anomalous nature of this subject's results argues toward the institution of an individually designed treatment program using a prolonged baseline as a means of discerning the appropriate contingencies for reinforcement.

Theta Suppression

Two of the five subjects were able to suppress theta occurrence during the treatment B_1 condition. The instability of this suppression is mirrored by the fact that neither of them had any transfer to either the pretreatment B_1 phase or the posttreatment B_1 phase. It is well to view the general lack of success in the suppression of theta in view of the pre-/posttraining EEGs. Three of the four subjects who were successful in seizure abatement produced significant decreases in the 1.0-3.01 and 3.01-6.02 Hz band (#4, #5, #6). None of these subjects produced a significant change in the 6.02-9.10 Hz range. It is suggested that the abnormally large number of counts in the 1.0-6.02 Hz range to begin with as compared to posttraining constitutes an important normalizing of these subjects' EEG patterns. In effect, these subjects began with slower average frequencies than was anticipated by the fourth hypothesis. Their training lessened the occurrence of delta activity. It is likely that more extensive training would have had an eventual effect upon the higher theta range (6-9 Hz).

Design Critique and Suggestions for Improvement

In discussing the strengths and weaknesses of the design we must begin by realizing that the results taken as a whole indicate an acquisition of alpha without a similar diminution of theta.

From the standpoint of design the major weakness of this study lies in an insufficient extended baseline period of EEG recording. If these periods were sufficiently extended, one would expect a non-significant slope indicating a consistent response mode from the subject. Two of the five subjects displayed significantly negative slopes in alpha percent and had this trend been allowed to continue may well have enabled the significance of the mean difference between baseline and contingent feedback (B_1) to be greater.

This statement is particularly important in regards to the mean differences between conditions. Given that the vast majority of the t-ratios for the differences between baseline and treatment conditions were significant, a stable baseline period may well have enhanced the possibility of significant differences in level, too.

One method of evaluating these changes from the baseline would be to employ a multiple baseline procedure. Essentially this entails a number of responses identified and measured over time to provide baselines against which changes can be evaluated. When the baselines are established, the experimenter then applies a treatment condition to one of the behaviors and observes little or no change in the other variable (Baer, Wolf and Risley, 1968). In this way significant results would be more readily determined through the contrast of concurrent baseline and treatment conditions.

Next, there appeared to be significant carryover effects with measures of the dependent variables. In some cases, seizure rate continued to remain depressed during the noncontingent conditions as it had during the contingent ones. There are two aspects of the carryover effect that must be explored: one, its relationship to learning, a positive aspect and, second, its duration indicating the need for a longer noncontingent period.

First, if the subject is being trained to respond under stimulus control, one would expect some form of generalization outside of the training situation. If the subject can only inhibit seizure production during training trials, this procedure would be of little utility in his everyday life. This carryover then can be considered a partially positive result because of the presumed learned nature of feedback contingencies.

Second, in attempting to demonstrate an effect one would assume that by preventing the connection between EEG production and the physiological feedback signal responding would return to baseline levels. This did not take place presumably because tne noncontingent condition was of insufficient duration to reestablish the baseline levels. Yet, the only method of determining the permanence of the changed level would be to utilize a prolonged noncontingent feedback phase.

Lastly, in dealing with noncontingent manipulations in seizure reduction, the ethical dilemma arises of reintroducing seizure activity where it may have attenuated. In dealing with human subjects who experience the debilitating effects of seizures, one sees a clash between the very real necessities of scientific inquiry and the best interests of the subject.

Why was alpha activity significantly controlled and not theta? From the standpoint of task complexity both alpha acquisition and theta suppression can be confidently considered fairly simple discrimination tasks involving markedly different stimuli. It would seem that it is easier to enhance an ongoing response set than to inhibit it. In this dual task we are looking at the acquisition and recognition of a stimulus and the subject's response. These are two very different tasks; the recognition of a stimulus may be far easier than actually responding appropriately. Response differentiation could have been facilitated by reinforcing each response individually over time and then combining feedback signals.

Suggestions for Future Research

The aim of the present study was two-fold: one, to evaluate the proposition that feedback of midrange EEG frequencies of the EEG via synchronous alpha wave enhancement and theta wave suppression was an efficacious means of seizure control; two, to attempt to develop a procedure that was clinically viable in the sense of being able to diminish the time needed to train an individual to attenuate seizure rate.

In any suggestions for future research the aforementioned methodological considerations are prominent and provide many of the suggestions for part one of this section.

In devising future studies, one method of deciding upon the most efficacious dependent variables is by correlating seizure diary information rate with EEG parameters over a prolonged baseline. When this is done, one would select only those EEG variables for reinforcement which are positively related with seizure rate. This might well

permit the optimal choice of EEG variables for an individual subject which, when reinforced or suppressed, would lead to seizure diminution. This procedure would lend itself to an empirical evaluation of EEG parameters, which when modified would lead to normalizing the EEG of epileptics.

Certainly an important covariant in a study of this nature is the subject's present and prior history of adherence to an anticonvulsant regimen. In this regard, it is suggested that along with a prolonged baseline of EEG and seizure diary data anticonvulsant blood levels be included prior to and throughout the course of training. Richens (1976) has indicated that the very fact of telling subjects that you are able to monitor the level of medication changes their rate of compliance in a positive direction. If, during the course of physiological feedback training, it is seen that seizures are beginning to attenuate, it would be well to monitor these levels closely to see if there has been any change due to increased compliance. In effect, what is argued for is a multidisciplinary approach that combines the medical approach of diagnosis and anticonvulsant therapy with that of the behavioral, which seeks to modify the ability of the individual to become aware of and control pertinent physiological states that are antithetical to seizure production.

In developing a consistent, long lasting program for seizure control, the role of home feedback equipment is an important adjunct. Sterman (1978) utilized such devices and 4-channel strip chart recorders which kept track of the time of each session, its duration, and the number of rewards obtained. As the advances of technology make this a more cost effective approach, the benefit to the subject

and investigator is great. The subject has a means of training available to him that increases the ease and possibility of compliance. For the investigator data are collected in the subject's natural environment, and the problem of generalization from laboratory to home is considerably lessened.

Another important subject covariant is that of expectancy of success. Each of the subjects had experienced a prolonged and chronic illness which modified their life in significant ways. Stroebel and Glueck (1973), in discussing the expectancy or placebo effects common to all therapies, contends that individuals who acquire and maintain a realistic set of expectancies (basically, one that views the procedure as possibly beneficial within the constraints of what is possible) will have a greater chance of success than those who either over- or undervalue the possible outcome of the procedure. Stroebel and Glueck (1973) consider simple, well presented information as the backbone of a realistic set of expectancies. It is incumbent upon any research in this area to devise a means of gauging the subject's initial knowledge and attitudes about the disorder and the training procedure and, then, to attempt to increase realistic expectancy by a systematic program to educate and inform him in a step-by-step fashion. During this procedure it is important to have an ongoing method to measure expectancy so as to gauge the effectiveness of the training.

APPENDIX A
TRAINING TAPE SCRIPT

As examples of what you may expect to hear, short excerpts, previously recorded, will be played for you.

First, a tone stays on 20 percent of the time. You will notice the tone varies both in loudness and pitch and goes on and off frequently.

(tape here)

Next, a tone with 50 percent success. Notice also that the tone varies in loudness and pitch and stays on about 50 percent of the time.

(tape here)

Now, a tone that stays on 100 percent of the time. Here the tone stays on nearly all of the time but slightly varies in terms of pitch and loudness.

(tape here)

If you become sleepy or produce slow waves associated with seizures, an alarm will sound.

(tape here)

This alarm will remain on as long as you are producing these slow waves. Your job is to keep the alarm off and the sound on.

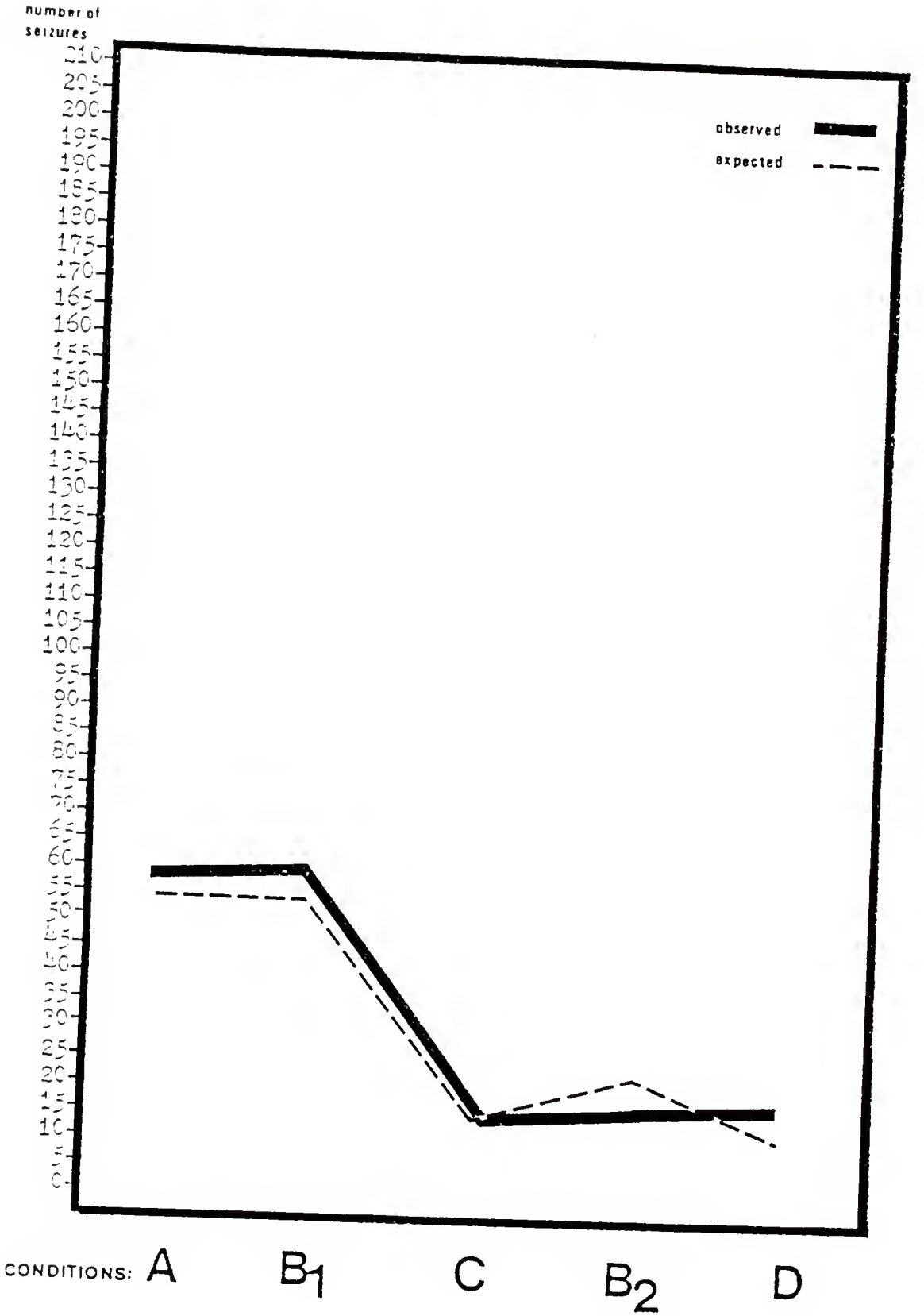
If you move around very much, a crackling sound called artifact will be heard.

(tape here)

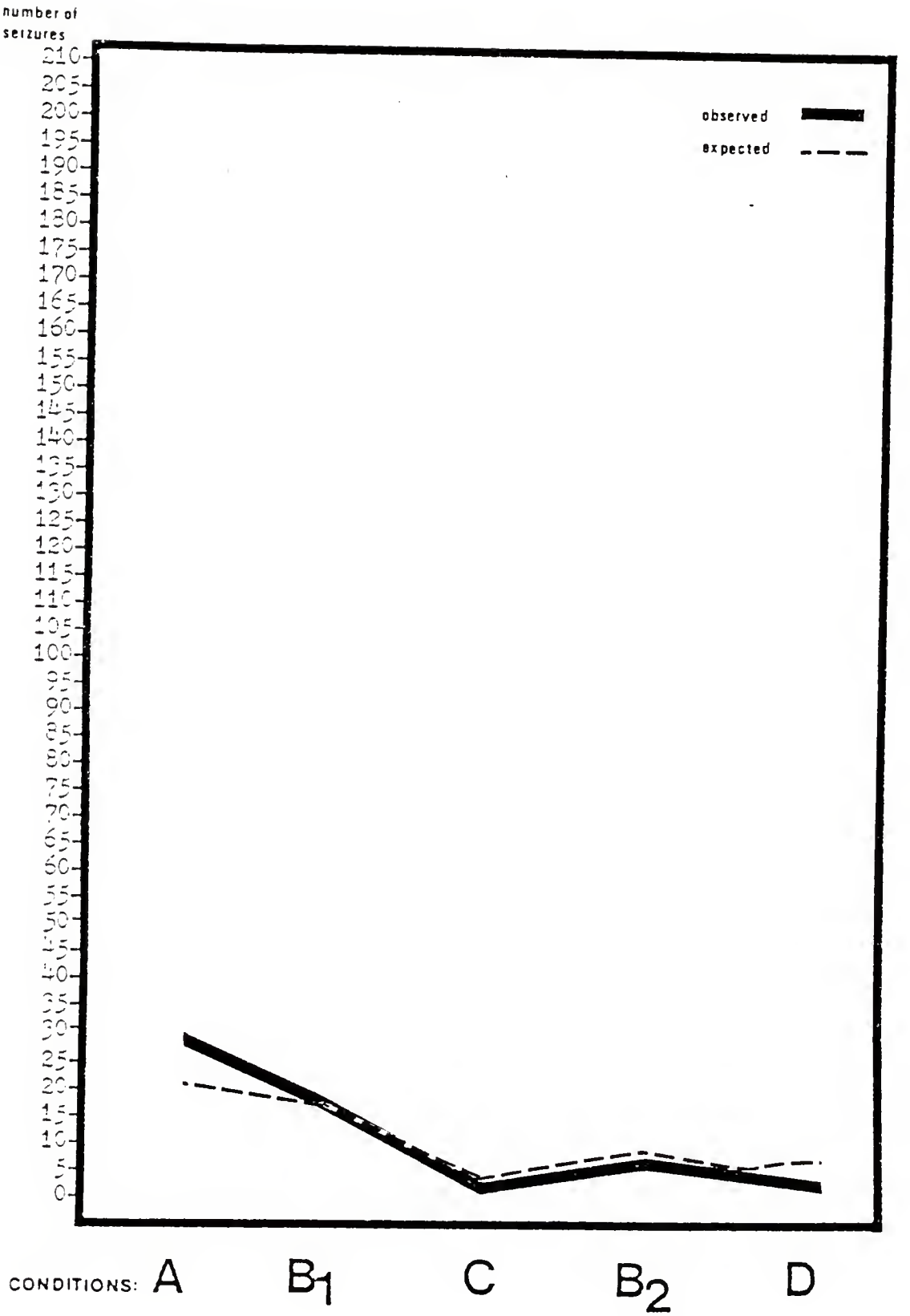
Try to remain as still as possible so as not to produce this dound.

Again, try not to become sleepy and work at keeping the sound on and the alarm off. This session will begin now.

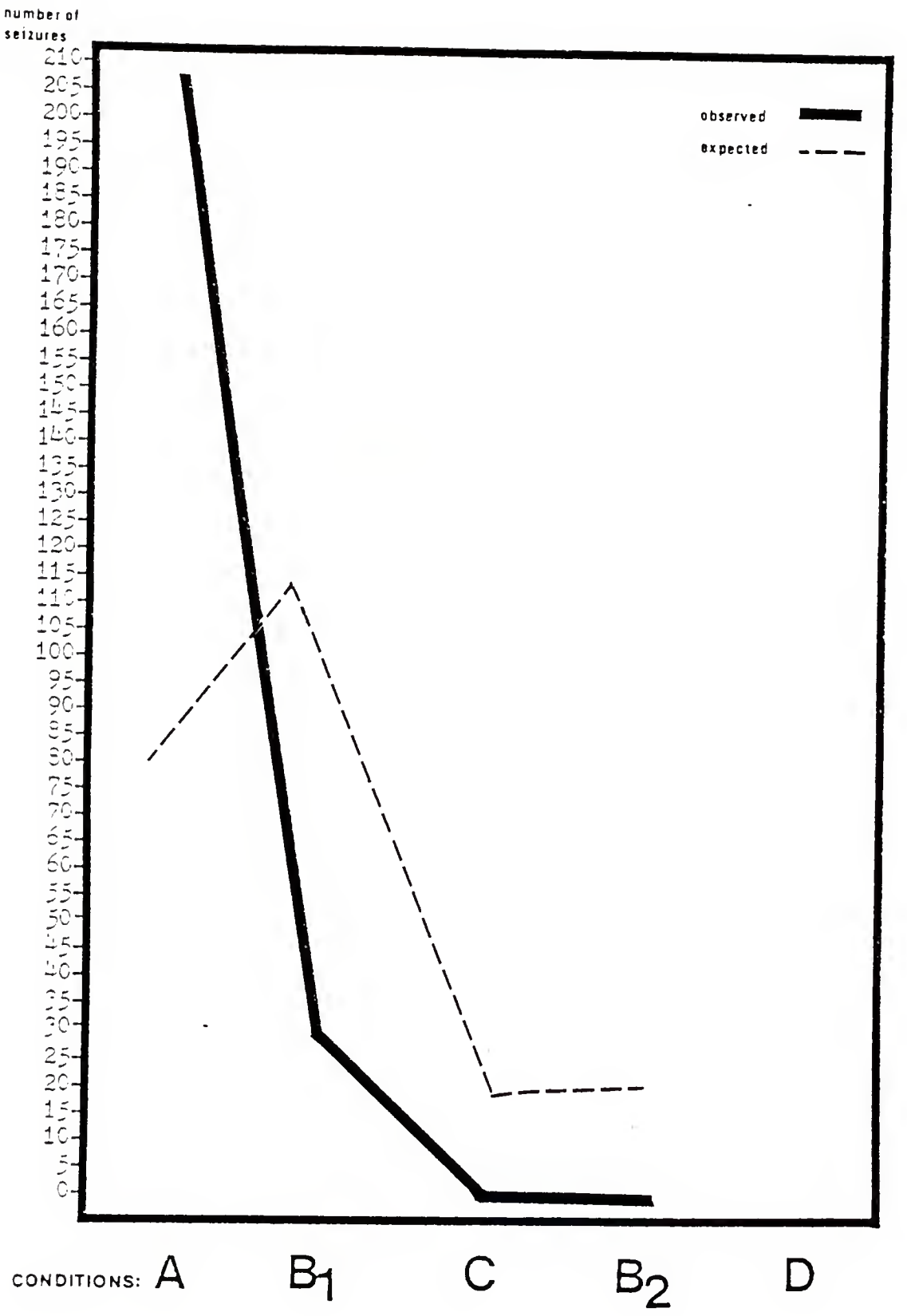
APPENDIX B
CHI SQUARE PLOT OF OBSERVED VS. EXPECTED SEIZURE FREQUENCIES BY CONDITION



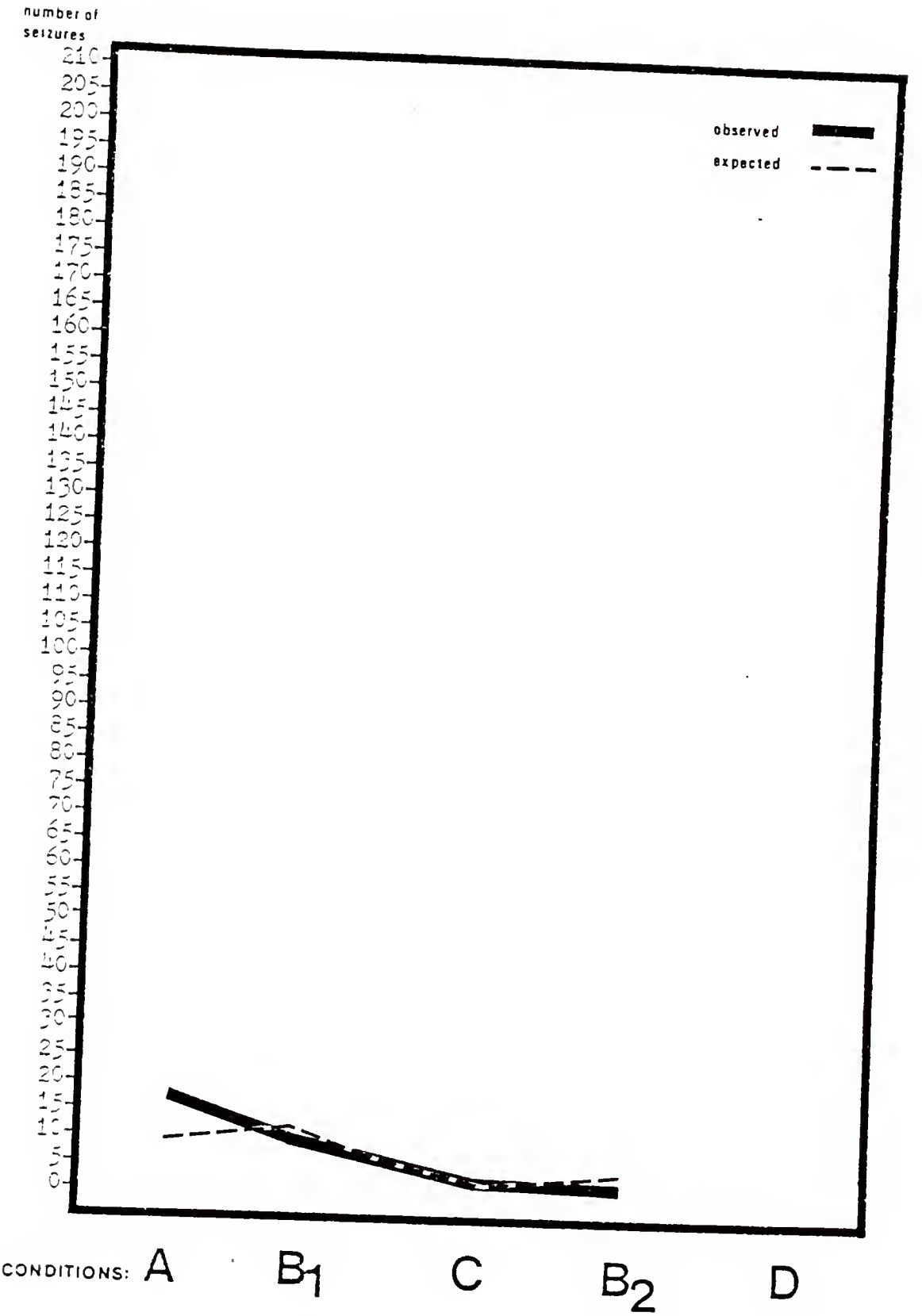
Chi Square Plot of Observed vs. Expected Seizure Frequencies
by Condition for Subject #1



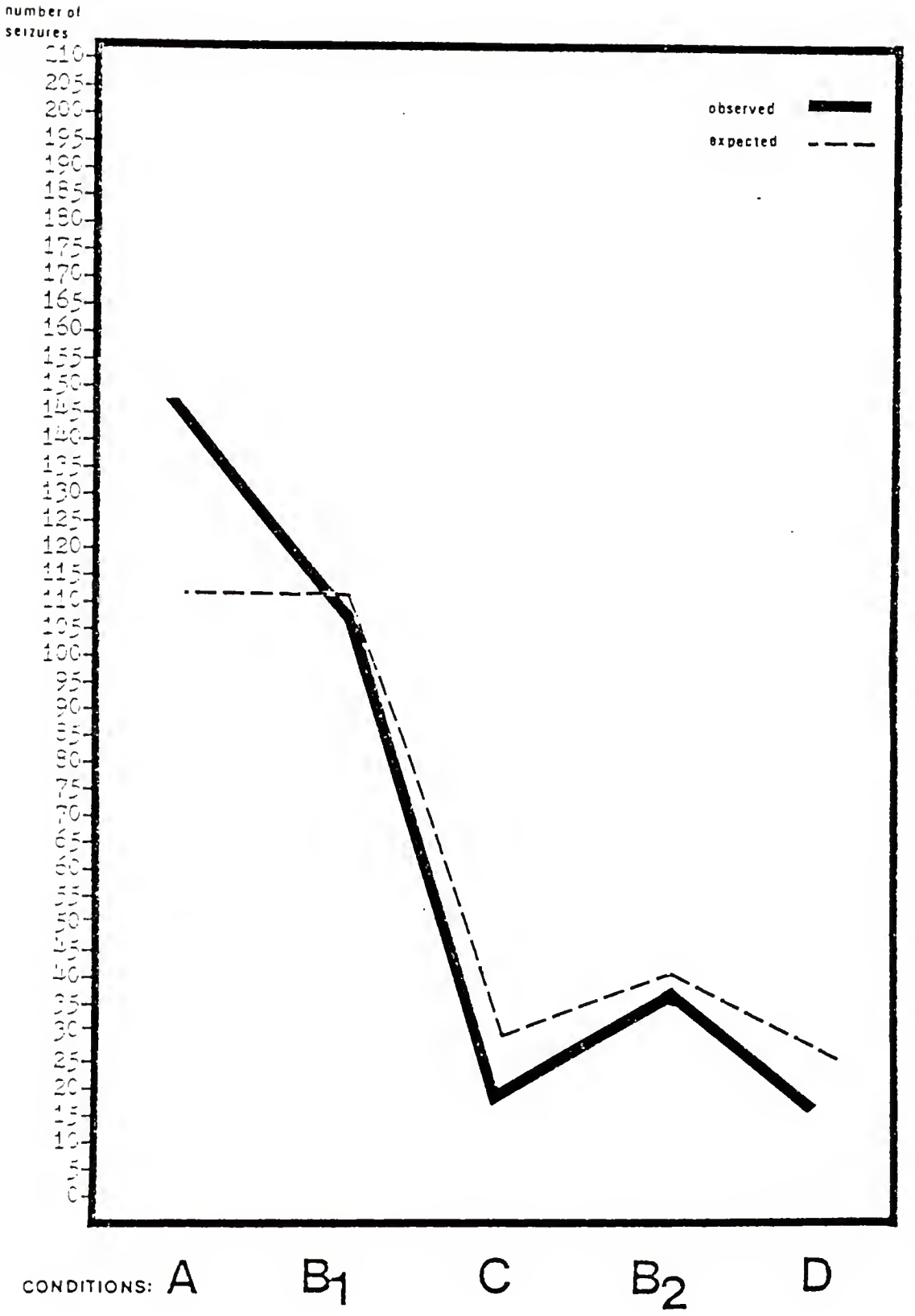
Chi Square Plot of Observed vs. Expected Seizure Frequencies by Condition for Subject #2



Chi Square Plot of Observed vs. Expected Seizure Frequencies by Condition for Subject #4



Chi Square Plot of Observed vs. Expected Seizure Frequencies by Condition for Subject #5



Chi Square Plot of Observed vs. Expected Seizure Frequencies
by Condition for Subject #6

APPENDIX C
SUMMARY TABLE OF SIGNIFICANT SLOPES

Condition	Subjects				
	1	2	4	5	6
A (Baseline)					
Alpha Percent (APR)	NS	6.988****	-4.867**	NS	-2.118*
Average Frequency (AFQ)	NS	-0.547****	NS	NS	NS
Average Amplitude (AAM)	NS	NS	NS	-0.964***	NS
Theta Percent (TPR)	4.055****	NS	NS	-1.857***	NS
Theta Count (TCO)	12.85****	NS	NS	-5.80****	NS
X AFQ VAR	NS	-0.196*	0.141***	NS	NS
X AAM VAR	NS	NS	NS	-17.73*	NS
B_1 (Pretreatment)					
APR	1.161*	0.491*	1.00*	NS	2.559***
AFQ	NS	-0.035*	NS	-0.062****	-0.112**
AAM	NS	NS	NS	-0.051*	-0.266*
TPR	NS	NS	NS	NS	NS
TCO	NS	NS	NS	NS	NS
X AFQ VAR	-0.017*	-0.014*	NS	NS	NS
X AAM VAR	NS	NS	NS	NS	NS
B_1 (Treatment)					
APR	0.794****	NS	0.575***	0.660****	0.715***
AFQ	NS	-0.055****	NS	-0.027***	-0.028**
AAM	-0.157****	NS	0.071**	0.053***	NS
TPR	-0.343**	NS	-0.115**	NS	NS
TCO	-1.036**	NS	-0.438**	NS	NS
X AFQ VAR	-0.022**	-0.008**	NS	0.009**	NS
X AAM VAR	NS	0.640*	1.025***	0.158*	NS

*p ≤ .05
**p ≤ .01
***P ≤ .001
****p ≤ .0001

Condition	Subjects				
	1	2	4	5	6
B_1 (Posttreatment)					
APR	NS	NS	0.957**	0.940**	NS
AFQ	NS	NS	-0.032**	-0.045*	NS
AAM	NS	NS	NS	NS	-0.156***
TPR	NS	NS	NS	NS	NS
TCO	NS	NS	NS	NS	NS
X AFQ VAR	NS	-0.017**	NS	NS	NS
X AAM VAR	NS	NS	0.482*	NS	NS
C (Pre-noncontingent)					
APR	-15.12*	NS	NS	NS	NS
AFQ	NS	NS	NS	NS	NS
AAM	NS	NS	-0.432*	NS	NS
TPR	NS	NS	NS	NS	NS
TCO	NS	NS	NS	1.689*	NS
X AFQ VAR	NS	NS	0.057*	NS	NS
X AAM VAR	NS	NS	NS	NS	NS
C (Noncontingent feedback)					
APR	-7.318****	NS	NS	-0.556*	1.676*
AFQ	0.611****	NS	0.106*	0.109**	NS
AAM	0.882****	NS	NS	-0.469****	NS
TPR	NS	NS	NS	-1.428**	NS
TCO	NS	NS	NS	-2.281*	NS
X AFQ VAR	NS	NS	NS	-0.055****	-0.117*
X AAM VAR	NS	NS	-5.382***	-8.148****	NS

Condition	Subjects				
	1	2	4	5	6
C (Post-noncontingent)					
APR	NS	NS	-5.869*	NS	NS
AFQ	0.566*	NS	0.164*	NS	0.114*
AAM	NS	NS	0.538*	NS	1.236*
TPR	NS	NS	NS	NS	NS
TCO	NS	NS	NS	NS	NS
X AFQ VAR	NS	NS	NS	NS	NS
X AAM VAR	NS	NS	NS	NS	NS
B_2 (Pretreatment)					
APR	NS	NS	NS	NS	NS
AFO	NS	NS	-0.105*	NS	NS
AAM	NS	NS	NS	NS	0.625*
TPR	-1.825*	NS	2.159*	NS	NS
TCO	-5.684*	-3.023**	6.849*	NS	NS
X AFQ VAR	NS	NS	NS	NS	NS
X AAM VAR	NS	NS	NS	-0.661*	1.468*
B_2 (Treatment)					
APR	NS	NS	NS	-0.937****	-0.858**
AFQ	-0.135***	NS	0.106*	-0.072****	NS
AAM	-0.303****	0.242*	NS	NS	NS
TPR	-0.405****	NS	NS	-0.641****	NS
TCO	-1.685****	NS	NS	-2.840****	NS
X AFQ VAR	NS	NS	NS	NS	NS
X AAM VAR	NS	NS	-5.382***	NS	NS

Condition	Subjects				
	1	2	4	5	6
B_2 (Posttreatment)					
APR	NS	NS	NS	NS	NS
AFQ	NS	NS	-0.116**	NS	0.138**
AAM	NS	NS	0.940*	NS	NS
TPR	NS	-1.449*	NS	NS	-4.967*
TCO	NS	NS	-1.546*	NS	NS
X AFQ VAR	NS	NS	0.173*	NS	NS
X AAM VAR	NS	NS	6.122*	NS	NS
D (Pretheta)					
APR	NS	NS			NS
AFO	NS	NS			NS
AAM	NS	NS			NS
TPR	NS	NS			NS
TCO	NS	NS			NS
X AFQ VAR	NS	NS			NS
X AAM VAR	NS	NS			NS
D (Theta Suppression)					
APR	-8.728****	NS			NS
AFQ	0.321****	NS			-0.134**
AAM	0.882****	0.242*			NS
TPR	-0.358****	NS			-0.617****
TCO	NS	NS			-2.765****
X AFQ VAR	NS	NS			NS
X AAM VAR	NS	NS			3.006**

Condition	Subjects				
	1	2	4	5	6
D (Posttheta)					
APR	NS	NS			NS
AFQ	NS	NS			NS
AAM	0.637*	NS			1.069*
TPR	NS	NS			NS
TCO	NS	NS			NS
X AFQ VAR	NS	NS			NS
X AAM VAR	NS	NS			NS

APPENDIX D
SELECTED PAIR-WISE CONTRASTS BETWEEN BASELINE AND TREATMENT CONDITIONS FOR SLOPES AND LEVELS

Selected Pair-wise Contrasts Between Baseline and Treatment Conditions for Slopes and Levels During the Alpha Pretreatment Period

Subject	Pairs			
	$A\text{-}B_1$	A-C	$A\text{-}B_2$	A-D
01				
t-ratio for slopes	-3.86*	17.78**	-2.86	3.64*
t-ratio for levels	0.79	0.34	-0.07	0.49
df	45	32	35	32
02				
t-ratio for slopes	32.78**	11.22**	22.86**	13.37**
t-ratio for levels	-0.98	-0.43	-0.68	-0.96
df	45	32	35	32
04				
t-ratio for slopes	-15.17**	-9.27**	-8.44**	x
t-ratio for levels	-0.06	-0.52	-0.04	x
df	46	32	34	x
05				
t-ratio for slopes	-3.43	0.17	-7.56**	x
t-ratio for levels	-0.68	-0.36	-0.94	x
df	46	32	34	x
06				
t-ratio for slopes	-19.47*	-4.48**	-3.49*	-4.80**
t-ratio for levels	-0.01	-0.49	-0.83	-0.77
df	46	32	34	33

*$p \leq .05$
**$p \leq .01$

Selected Pair-wise Contrasts Between Baseline and Treatment Conditions for Slopes and Levels During the Alpha Treatment Period

Subject	Pairs			
	$A\text{-}B_1$	A-C	$A\text{-}B_2$	A-D
01				
t-ratio for slopes	-7.69**	27.92**	-0.91	32.22**
t-ratio for levels	-0.65	0.57	-2.19	1.80
df	165	60	84	60
02				
t-ratio for slopes	97.23**	37.45**	57.43**	52.04**
t-ratio for levels	-1.26	-1.18	-2.65	-2.78
df	163	61	84	60
04				
t-ratio for slopes	-40.36**	-10.73**	-12.84**	x
t-ratio for levels	-0.72	0.80	-1.63	x
df	172	60	76	x
05				
t-ratio for slopes	-14.64**	2.66	6.99**	x
t-ratio for levels	-1.29	-1.18	-2.70	x
df	172	60	76	x
06				
t-ratio for slopes	-34.15**	-17.59**	-8.83**	-10.25**
t-ratio for levels	-0.79	-0.83	-2.48	-1.12
df	172	60	76	68

*$p \leq .05$
**$p \leq .01$

Selected Pair-wise Contrasts Between Baseline and Treatment Conditions for Slopes and Levels During the Alpha Posttreatment Period

Subject	Pairs			
	$A\text{-}B_1$	A-C	$A\text{-}B_2$	A-D
01				
t-ratio for slopes	-3.22	0.06	-1.56	13.09**
t-ratio for levels	0.13	0.15	-0.51	1.13
df	45	32	35	32
02				
t-ratio for slopes	35.56**	21.18**	17.47**	20.43**
t-ratio for levels	-0.66	-0.50	-0.60	-0.70
df	45	32	35	32
04				
t-ratio for slopes	-15.22**	1.20	-6.04**	x
t-ratio for levels	-0.36	-0.16	0.09	x
df	46	32	34	x
05				
t-ratio for slopes	-6.83**	-1.13	6.39**	x
t-ratio for levels	-0.83	-0.74	-0.14	x
df	46	32	34	x
06				
t-ratio for slopes	-12.46**	-3.81**	-8.36**	-8.86**
t-ratio for levels	-0.95	-0.67	-0.81	-0.36
df	46	32	34	33

*$p \leq .05$
**$p \leq .01$

Selected Pair-wise Contrasts Between Baseline and Treatment Conditions for Slopes and Levels During the Theta Pretreatment Period

Subject	Pairs			
	$A\text{-}B_1$	A-C	$A\text{-}B_2$	A-D
01				
t-ratio for slopes	50.96**	5.59**	39.81**	12.46**
t-ratio for levels	-0.07	0.16	-0.30	0.04
df	45	32	35	32
02				
t-ratio for slopes	-3.56*	0.52	-3.55*	-5.43**
t-ratio for levels	-0.28	-0.83	-0.04	-0.21
df	45	32	35	32
04				
t-ratio for slopes	0.78	1.23	-3.80**	x
t-ratio for levels	0.16	0.08	0.80	x
df	46	32	34	x
05				
t-ratio for slopes	-19.60**	-2.84	-9.86**	x
t-ratio for levels	-1.64	-4.05**	-3.12	x
df	46	32	34	x
06				
t-ratio for slopes	2.38	-8.33**	-0.49	-0.62
t-ratio for levels	-0.57	-0.84	-1.27	-0.03
df	46	32	34	33

*$p \leq .05$
**$p \leq .01$

Selected Pair-wise Contrasts Between Baseline and Treatment Conditions for Slopes and Levels During the Theta Treatment Period

Subject	Pairs			
	$A\text{-}B_1$	A-C	$A\text{-}B_2$	A-D
01				
t-ratio for slopes	139.61**	19.46**	107.49**	82.77**
t-ratio for levels	0.11	-1.00	1.61	1.54
df	165	60	84	60
02				
t-ratio for slopes	-8.56**	-7.51**	-4.23**	5.93**
t-ratio for levels	0.69	-1.24	2.99*	2.87
df	163	61	84	60
04				
t-ratio for slopes	5.29**	2.86	4.01**	x
t-ratio for levels	-0.19	-1.31	0.21	x
df	172	60	76	x
05				
t-ratio for slopes	-46.48**	-3.75*	-17.47**	x
t-ratio for levels	-0.08	-1.82	1.24	x
df	172	60	76	x
06				
t-ratio for slopes	4.55**	-0.75	5.73**	8.83**
t-ratio for levels	0.20	-1.39	0.63	1.90
df	172	60	76	68

*$p \leq .05$
**$p \leq .01$

Selected Pair-wise Contrasts Between Baseline and Treatment Conditions for Slopes and Levels During the Theta Posttreatment Period

Subject	Pairs			
	$A\text{-}B_1$	A-C	$A\text{-}B_2$	A-D
01				
t-ratio for slopes	48.13**	7.58**	34.09**	16.44**
t-ratio for levels	-0.31	-0.49	-0.16	-0.18
df	45	32	35	32
02				
t-ratio for slopes	-2.11	-2.21	3.15	-1.48
t-ratio for levels	-0.33	-0.52	0.40	0.24
df	45	32	35	32
04				
t-ratio for slopes	.84	1.22	1.65	x
t-ratio for levels	-0.69	-0.77	-0.46	x
df	46	32	34	x
05				
t-ratio for slopes	-19.29**	-6.32**	-7.75**	x
t-ratio for levels	-0.46	-1.09	0.07	x
df	46	32	34	x
06				
t-ratio for slopes	3.82**	-7.22**	12.73**	1.32
t-ratio for levels	-0.74	-1.58	-0.56	-0.04
df	46	32	34	33

*$p \leq .05$
**$p \leq .01$

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I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Hugh C. Davis, Chairman
Professor of Clinical Psychology

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

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